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AN ENQUIRY INTO THE ETIOLOGY, MANIFESTATIONS AND PREVENTION OF PNEUMONIA AMONGST NATIVES ON THE RAND, RECRUITED FROM TROPICAL AREAS.

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"Wherefore I have not thought that it stood in need of an empty hypothesis like those subjects which are occult and dubious, in attempting to handle which it is necessary to use some hypothesis; as for example, with regard to things above us and things below the earth; if anyone should treat of these and undertake to declare how they are constituted, the reader or hearer could not find out whether what is delivered be true or false; for there is nothing which can be referred to in order to discover the truth."

Hippocrates—"On Ancient Medicine"

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AN ENQUIRY INTO THE ETIOLOGY, MANIFESTATIONS, AND PREVENTION OF PNEUMONIA AMONGST NATIVES ON THE RAND RECRUITED FROM "TROPICAL AREAS."

By G. D. MAYNARD, F.R.C.S.E.

In any discussion of the disease known as "Pneumonia" it is necessary at the outset to define clearly what is meant by this term. A few years ago a clear cut distinction was supposed to exist between lobar and catarrhal pneumonia. With the growth of bacteriology a tendency has arisen to class as pneumonic infections, all diseases which appear to be causally associated with the pneumococcus of Fraenkel. But, unfortunately, the term pneumonia is associated with a definite group of symptoms and physical signs, and such symptoms and signs *may* occur unassociated with the pneumococcus, while, on the other hand, this organism is frequently present in diseases which do not show the classical symptoms. If we attempt to record under the heading "Pneumonia" all diseases causally associated with the pneumococcus we are faced by grave, if not insurmountable, clinical difficulties; whereas, no survey of the effects of the pneumococcus can be considered complete which takes into account only one form of its manifestations.

Among the Natives recruited from tropical areas, who supply the bulk of the matter dealt with in this paper, Fraenkel's pneumococcus was frequently obtained in pure culture, by lung-puncture, from cases of lobar pneumonia. This organism has been also obtained from the sputum, and by lung-puncture, from some cases of broncho pneumonia. Further, it has been recovered in pure culture from abscesses unassociated with any lung symptoms, for example from a suppurating knee joint; and repeatedly from the cerebro-spinal-fluid. Finally, the pneumococcus was not infrequently recovered from the blood of patients suffering from lobar pneumonia. These observations show that amongst these Natives pneumococcal infections are as protean in their manifestations as they are known to be amongst Europeans.

The term "Pneumonia" will be used throughout this paper to mean the disease clinically known as lobar pneumonia when bacteriologically associated with the diplococcus of Fraenkel. We must not, however, lose sight of the fact that a survey of Pneumonia will be only a partial survey of the effects of the pneumococcus; but in the absence of a complete bacteriological examination in all cases of disease it is a limitation necessitated by our ignorance.

This paper is divided into the following sections:—

1. A consideration of the prevalence of pneumonia as influenced by race and country of birth. Death and attack rates, the case mortality, and the influence of season.
2. The clinical manifestations, course, symptoms, and gross morbid anatomy of the disease.
3. A consideration as to whether there be any evidence that the disease is infectious.
4. A consideration of the effect of prophylactic inoculation of a pneumococcal vaccine on the incidence and mortality of pneumonia.
5. General conclusions.

SECTION I.

A discussion of the prevalence of pneumonia as influenced by race and country of birth. Death and attack rates, the case mortality, and the influence of season.

Some of the Natives working on the mines of the Witwatersrand are recruited from districts north of lattitude 22° S. These Natives are colloquially termed "Tropical Natives"; this convenient expression we shall continue to use. The majority come from the Portuguese districts of Quilimane, Mozambique, Portuguese Nyassaland, and the district of Tete. It is impossible to divide these Natives into tribes. In recent years so much intermarriage has taken place that, except on very broad lines, tribal distinctions are not maintained. There are marked differences, however, between the Natives in the different territories in regard to their physique and liability to disease. These differences necessitate a separate consideration of the groups. Natives from some of these districts are apparently more heterogeneous than others, and this is particularly marked amongst the Quilimane Natives. For some purposes, therefore, they have been divided into four groups according to geographical sub-divisions of the district. All "Tropical Natives" employed on the Rand are recruited by the Witwatersrand Native Labour Association, and it is owing to the courtesy of this Association that its valuable records have been placed at my disposal.

The Natives are recruited at their kraals, and marched from rest-camp to rest-camp until they reach a depôt at the port of embarkation. At these depôts they are housed and fed while awaiting the steamer which is to take them to Delagoa Bay. The ports of embarkation from North to South are as follows:—Port Amelia in Nyassaland, Mozambique, Angoche, Pebane for the district of Quilimane, and Chinde where the Tete Natives and Protectorate Independent Territorial Natives (the latter subsequently referred to as P.I.T.) are shipped. These Natives arrive at Chinde by boat viâ the Zambezi river. The voyage to Lourenço Marques occupies from five to twelve days. On arrival here the Natives are transferred to the train, and sent to the central low veld station of the Company at Ressano Garcia, where they are medically examined, detained for a few days, and then if passed as fit for mine work forwarded to the Rand. If the Medical Officer is not satisfied with their condition they are either sent home, or further detained at this place, to enable them to recuperate in a district with a climate similar to that of their homes. On their arrival in Johannesburg they are sent to the Association's compound, where they remain under close medical supervision for a further period of at least three weeks.* At the end of this time they are again medically examined, and if deemed fit for work are allotted to the mines, but if found to be unfit they are either returned to their homes or detained for a further period. It is during their stay in the Johannesburg compound that most of the records dealt with in this paper have been obtained.

The meaning of the terms used in connection with these Natives will now be readily understood. A Native not allotted to a mine at the end of three weeks with the gang to which he belongs is called a "detention"; if he be sent home a "reject." It is considered inadvisable to bring Natives from tropical districts on to the Rand during the winter, and, therefore, no recruiting is carried on in these areas during the four months April to July. The following tables, I. and Ia., show the numbers recruited from each area, with the repatriations, detentions, and deaths in the Johannesburg compound. The figures are obtained from the total number of Natives arriving in the seasons 1910 to 1911, and 1911 to 1912, and one-half of the recruiting season 1912 to 1913, that is, from August to 31st December, 1912.

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* "Tropical Natives" are not permitted to work underground for a period of four weeks from the time of their arrival. There is no restriction to their doing surface work earlier, hence the period in the compound is on the average about three weeks.

TABLE I.

Plain figures give actual numbers, italics rates per 1000.

Territorial origin.		Season.	Number of Natives recruited.	Detentions.	Repatriations.	Deaths in compound.
Mozambique	...	1910-11	5047	1368 *271·1*	174 *34·5*	220 *43·6*
,,	...	1911-12	4812	932 *193·7*	172 *35·7*	168 *34·9*
,,	...	1912	3412	637 *186·7*	71 *20·8*	129 *37·8*
,,	...	Total	13271	2937 *221·3*	417 *31·4*	517 *39·0*
Angoche	...	1910-11	3636	929 *255·5*	77 *21·2*	191 *52·5*
,,	...	1911-12	2517	518 *205·8*	362 *14·4*	161 *64·0*
,,	...	1912	1180	285 *241·5*	13 *11·0*	53 *44·9*
,,	...	Total	7333	1732 *236·1*	452 *61·6*	405 *55·2*
Quilimane	...	1910-11	2746	776 *282·6*	90 *32·8*	99 *36·1*
,,	...	1911-12	2106	467 *221·7*	90 *42·7*	58 *27·5*
,,	...	1912	1272	293 *230·3*	22 *17·3*	40 *31·4*
,,	...	Total	6124	1536 *250·8*	202 *33·0*	197 *32·2*

Territorial origin.	Season.	Number of natives recruited.	Detention.	Repatriation.	Deaths in compound.
Portuguese Nyassaland	1910-11	3574	652	61	85
			182·4	*17·1*	*23·8*
,,	1911-12	2639	350	62	61
			132·6	*23·5*	*23·1*
,,	1912	2808	454	29	78
			161·7	*10·3*	*27·8*
,,	Total	9021	1456	152	224
			161·4	*16·8*	*24·8*
Tete	1910-11	3077	315	19	16
			102·4	*6·2*	*5·2*
,,	1911-12	1409	177	15	20
			125·7	*10·7*	*14·2*
,,	1912	1585	232	3	11
			146·4	*1·9*	*6·9*
,,	Total	6071	724	37	47
			119·2	*6·1*	*7·7*
P.I.T.	1910-11	820	62	3	6
			75·6	*3·7*	*7·3*
,,	1911-12	1221	129	10	23
			105·7	*8·2*	*18·8*
,,	1912	200	36	1	4
			180·0	*5·0*	*20·0*
,,	Total	2241	227	14	33
			101·3	*6·2*	*14·7*
British Nyassaland Protectorate (B.N.P.)	1910-11	291	47	3	4
			161·5	*10·3*	*13·7*
	1911-12	938	158	9	22
			168·4	*9·6*	*23·5*
,,	1912	1			
,,	Total	1230	205	12	26
			166·7	*9·8*	*21·1*

TABLE Ia.

SUMMARY.

Rates per 1000 in italics.

Territorial origin	Number of Natives recruited.	Detention.	Repatriation.	Deaths in compound.
Mozambique ...	13271	2937	417	517
		221·3	*31·4*	*39·0*
Angoche ...	7333	1732	452	405
		236·1	*61·6*	*55·2*
Portuguese Nyassaland ...	9021	1456	152	224
		161·4	*16·8*	*24·8*
Quilimane ...	6124	1536	202	197
		250·8	*33·0*	*32·2*
Maganja da Costa ...	2950	548	69	54
		185·3	*23·3*	*18·3*
Lomwe ...	1620	590	60	74
		364·2	*37·0*	*45·7*
Higher M'Locwe ...	1042	301	46	51
		288·9	*44·1*	*49·0*
Lower M'Locwe ...	512	97	27	18
		189·5	*52·7*	*35·2*
P.I.T. ...	2241	227	14	33
		101·3	*6·2*	*14·7*
Tete ...	6071	724	37	47
		119·2	*6·1*	*7·7*
B.N.P. ...	1230	205	12	26
		166·7	*9·8*	*21·1*
Total ...	45291	8817	1286	1449
		194·7	*28·4*	*32·0*

It will be observed that the rates vary considerably with the areas from which these Natives are recruited; and there is, as we shall see later, a high correlation between the number of " detentions " and the number of deaths.

The effect of dividing the Quilimane Natives into sub-groups is instructive. The death-rate for the whole group is 32·2 per thousand, which is practically the same as that found for the whole table. The rates in the sub-groups vary widely, Natives from the district of Maganja da Costa having a comparatively low death-rate, while those from the other districts have a much higher rate; so that the observed rate of 32 per thousand masks wide differences within the group.

Returning to Table IA., it will be seen that the Natives with the highest death-rates are recruited from the districts of Angoche, Higher M'Locwe*, and Lomwe*, whilst those from the district of Tete have a much lower rate than that of any other group. The differences in the general death-rates are, as we shall see, largely due to variations in the prevalence of pneumonia, and from 44 to 82 per cent. of the total death-rate is due to the mortality from this disease. Excluding the group " diarrhœal diseases," Cerebro-spinal Meningitis is second to pneumonia in importance as a cause of death during the period of residence of these Natives in the W.N.L.A. Compound in Johannesburg. For this reason, and because its relative prevalence according to the territorial distribution of the Natives is not the same as that for pneumonia, the statistics of this disease have been included in Table II.

It is necessary to digress for a moment to enquire how far the death-rate from pneumonia may be regarded as an index to the attack-rate; that is, does a high death-rate from pneumonia necessarily indicate a high attack-rate? Clearly, if the case mortality were constant we could instantly deduce the one rate from the other; or given the death-rate, we could place the groups in the order of their liability to attack. In Column (10) of Table II. we see that the case mortality is not constant, and we therefore must determine whether a high death-rate is, or is not, associated with a high attack-rate. As the correlation of rates is unsatisfactory owing to a spurious correlation being thereby introduced, it is necessary to calculate the partial correlation by Professor Pearson's formula, which is as follows:—

If p = the population of the group, r = the number who recovered, and d = the number who died, then the correlation of recoveries and deaths for a constant population is:—

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* It is worth recording that the head Recruiter reports that in these districts the Natives are exceedingly lazy, do very little cultivation, and are often, therefore, in a half-starved condition.

$$ {}_{p}r_{rd} = \frac{r_{rd} - r_{rp}\, r_{dp}}{\sqrt{1 - r_{rp}^2}\,\sqrt{1 - r_{dp}^2}} $$

In the case of pneumonia among the groups classified according to territorial distribution the value is:—

$$ {}_{p}r_{rd} = {\cdot}9045 \pm {\cdot}0388 $$

and there is, therefore, a high correlation between recoveries and deaths amongst those attacked with this disease. It will, consequently, make little difference whether we employ the attack-rate or the death-rate in estimating the prevalence of pneumonia among these groups. As in most enquiries the death-rate is the only factor obtainable this rate will be here used, since the results will be of more value for comparative purposes.

To return to the death-rate from pneumonia as given in Table II., and represented graphically in Map I. This map represents the rates occurring on the Rand amongst the Natives recruited from the different geographical areas, and does not refer to the conditions prevailing in the territories themselves. It is, of course, diagramatic, and if further sub-division of the Natives from the different districts had been possible the abrupt transition of the rates as shown would no doubt be considerably modified. That marked variations in the liability to this disease exist among the Natives recruited from these areas is plain, and it will be observed that the districts from which the highest rates are obtained is roughly a triangular area from Lake Chirua to the coast. The treatment of the Natives in regard to methods of recruiting, housing, feeding, etc., is practically uniform, except that the varying distance of their homes from the coast necessitates in some cases a lengthy march to the port of embarkation. That this is not a critical factor, however, is shown by this map, for the areas which yield the worst death-rates are not those farthest from the coast. Speaking generally, it appears that the Natives from the inland districts are, when residing on the Rand, less prone to the disease than those from the coastal areas. In the United States, when allowances are made for the fact that pneumonia is more prevalent in urban than in rural districts, we find that persons residing in the coastal districts are less subject to this disease than those living inland.[1] In regard to these Natives we do not know whether in their homes a similar condition obtains, but if further investigation were to show that this be so, a possible explanation of the reversal of this condition on their arrival on the Rand would suggest itself; namely, that there had been a less rigorous selection amongst the Natives from the coastal areas.

Among other possible causes of these differences are:—

(1) That a general want of physique is correlated with a predisposition to all diseases.

(2) That certain tribes are particularly susceptible to pneumococcal infection, or

(3) That some factor favouring the spread of infection may be more marked in some groups than in others.

This last point will be dealt with more at length later, but it may be stated here that there is no evidence that a factor of infection has any influence in causing the variation in the death-rates. The Natives from the districts of Quilimane and Angoche are, at any rate on arrival at Delagoa Bay, of distinctly inferior physique to those recruited from Nyassaland and Tete; and also inferior, but to a less marked degree, to those coming from Mozambique. But, if general physical inferiority were the whole explanation, then we might expect that the death-rate from other diseases would show the same territorial distribution as the pneumonia rate.

A similarly prepared representation of the death-rate on the Rand from Cerebro-spinal Meningitis is shown in Map II. The figures used in preparing the maps, as well as those from which the attack-rates for the two diseases are calculated, are given in Table II. on page 10.

Here we see that the Natives from the three districts of Quilimane which show the highest death-rates from pneumonia, are, in respect to Cerebro-spinal Meningitis, among the best. The fact, however, that the numbers in these groups are smaller, must not be overlooked. Excluding these two diseases, the territorial distribution of the death-rate for all medical diseases gives a picture very similar to that of pneumonia. It is possible then, that the distribution of Cerebro-spinal Meningitis may be due to some peculiarity in method of spread, or in a special constitutional liability, and that general want of physique may still explain the variation in the death-rates from other diseases. We have not, however, at the present time, sufficient evidence to enable any suggestions to be formulated except in a tentative manner; that a marked and persistent difference in the tribal rates does occur, is, nevertheless, established.

It may be remarked that while some authorities state that the weaklings of a community are most prone to contract pneumonia, others assert that it is the robust who are most often attacked. A similar divergency in opinion is found amongst the medical practitioners engaged in Native work. This conflict of opinion need

TABLE II.

Tropical Natives recruited by W.N.L.A. during the period August, 1910, to December, 1912, inclusive.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
				PNEUMONIA.								C. S. MENINGITIS.					
Territorial Origin.	Number of Recruits.	Total deaths.	Death-rate per thousand.	Cases.	Morbidity Rate per thousand.	Deaths.	Death-rate per thousand.	Per cent. of all deaths.	Case Mortality per cent.	Other deaths.	Death-rate per thousand.	Cases.	Morbidity rate per thousand.	Deaths.	Death-rate per thousand.	Case mortality per cent.	Death-rate per thousand minus Pn. & C.S. Men.
Portuguese Nyassaland ...	9,021	224	24·8	290	32·2	106	11·8	47	36·6	118	13·1	80	8·9	42	4·7	52·6	8·4
Mozambique ...	13,271	517	39·0	674	50·8	244	18·4	47	36.2	273	20·6	134	10·1	92	6·9	68·6	13·7
Angoche	7,333	405	55·2	405	55·2	179	24·4	44	44·2	226	30·8	111	15·1	86	11·7	72·5	19·1
Maganja da Costa	2,950	54	18·3	77	26·0	27	9·1	50	35·1	27	9·1	13	4·4	10	3·3	77·0	5·8
Lomwe	1,620	74	45·7	89	54·9	44	27·2	57	49·5	30	18·5	3	1·9	3	1·9	100·0	16·6
Higher M'Locwe	1,042	51	49·0	71	68·2	31	29·8	61	43·7	20	19·2	3	2·9	1	1·0	33·3	18·2
Lower ,, ...	512	18	35·2	29	56·6	9	17·6	50	31·0	9	17·6	2	3·9	1	2·0	50·0	15·6
P.I.T.	2,241	33	14·7	51	22·7	27	12·0	82	52·9	6	8·5	1	0·4	1	0·4	100·0	8·1
Tete	6,071	47	7·7	94	15·5	27	4·4	57	28·7	20	3·3	8	1·3	5	0·8	62·5	2·5
B.N.P.	1,230	26	21·1	29	23·6	15	12·2	58	51·7	11	8·9	1	0·8	1	0·8	100·0	8·2
Total	45,291	1,449	32·0	1,809	39·9	709	15·7	49	39·2	740	16·3	356	7·9	242	5·3	68·0	11·0

occasion no surprise if such views are obtained merely from impressions rather than from carefully kept records; in other words, when the so-called "experiential method "[2&3] replaces a scientific analysis of recorded facts.

Natives whose physical condition is poor are detained in the W.N.L.A. Compound beyond the average twenty-one day period, and, as before explained, are termed "detentions." If their condition is so bad, that, in the opinion of the medical officer, they are not likely to be fit for allotment to the mines within a reasonable time they are returned to their homes, and are termed "rejects." The physical condition of these Natives also varies from season to season, and at different times during the same season; the number of "detentions" and "rejects" from any group reflects, therefore, to a certain extent the condition of the whole group. By correlating either the general death-rate, or the pneumonia death-rate, with the number of "detentions" or "rejects," we shall obtain some measure of the association between the physical condition of the Natives and their liability to death.

Correlating all deaths (d) with "rejects" (n) for constant population (p), by the partial correlation formula, we find:—

$$ {}_{p}r_{dn} = \cdot 6549 \pm \cdot 0715 $$

and for all deaths and "detentions" (m),

$$ {}_{p}r_{dm} = \cdot 8343 \pm \cdot 0381 $$

Similarly the values for pneumonia deaths (e) and "rejects," and for ~~all~~ deaths and "detentions" are:—
pneumonia

$$ {}_{p}r_{en} = \cdot 7764 \pm \cdot 0497 $$
$$ {}_{p}r_{em} = \cdot 4990 \pm \cdot 0941 $$

Thus it is seen that in gangs where physically inferior "boys" preponderate both the total and the pneumonia death-rates are higher than where the reverse condition exists. Too much stress must not be placed on these results, as there is a considerable correlation between the "tribe" (or more correctly the Natives from a certain geographical district), and number of "detentions" or "rejects," so that we may be measuring the "tribal" liability rather than the physical condition of the gang; unless a difference in physique be the only factor of importance in this respect. The figures are of interest because they show that the physically inferior gangs have both a higher general death-rate, and a higher pneumonia death-rate, than those arriving in better condition; and the results, even interpreted on a racial basis, show that racial susceptibility is associated with a degree of physical unfitness. With the present incomplete data at our disposal it would be unwise to be dogmatic on this question, but there are distinct indications that the physically unfit are more prone to pneumonia.

I have elsewhere shown[4] that the prevalence of pneumonia—taking the death-rate as a measure of the prevalence—is greatest amongst Natives during their first month of residence on the Rand, and that thereafter it progressively diminishes. With our present figures we can consider this problem as it relates to new arrivals during their first few weeks of residence in Johannesburg. With the exception of loss by death, the population in the W.N.L.A. Compound is stationary for all "Tropical Natives" for approximately three weeks. Dividing the first nineteen days into five-day periods, the number attacked in each period out of a total of 45,291 Natives, is as follows:—

TABLE III.

Day after arrival.	Number attacked.	Attack rate per cent.
0— 4	526	1·16
5— 9	389	0·86
10—14	314	0·69
15—19	275	0·61

Here again we see that the period of greatest risk is that immediately following arrival, the greatest number of cases being admitted to hospital on the first day. Thus, the liability commences suddenly and immediately after arrival, and decreases subsequently from day to day. This distribution is shown in Chart I., as also the theoretical curve, which is of the "J_1" type. It will be observed that the start is within the first twenty-four hours. The actual number of cases recorded is greater in the second twenty-four-hour period, but this is due to the fact that the first period is not really one of twenty-four hours, because the first day as determined from the compound books is often a period of only twelve hours, or even less. The marked reduction in attack-rate with the length of residence is a fact which must not be overlooked when we consider the evidence as to the probability of case to case infection. That this fall continues after the Natives are removed to the mines is also shown by other figures at our disposal. In the experiment conducted with 8,123 Natives, which will be presently described, and is subsequently referred to as the "8000 experiment," the attack-rate month by month was recorded, and is here given in Table IV., and shown graphically on Chart II.

TABLE IV

Month after arrival.	Number attacked.	Attack rate per cent.
0	312	3·89
1	203	2·58
2	155	2·00
3	139	1·83
4	125	1·68
5	89	1·22

This Table also shows that amongst Natives recruited in "tropical areas" the maximum attack-rate occurs immediately on their arrival on the Rand, and gradually falls as the period of residence increases. The influence of season in producing variations in the attack-rate does not in any way enter into the problem in regard to the figures given in Table III.; and it is probably not of importance in Table IV., because the Natives composing the population dealt with in this table were recruited over a period from August to the middle of November, that is, from the coldest time during which recruiting from these districts is undertaken, up to, and well into the warm season. Chart III. shows the daily admissions from Cerebro-spinal Meningitis; and it is inserted here to show that this distribution is not similar in type to that found for pneumonia. There are some special features in this distribution which need not detain us here, but the distributions are clearly of different types.

It is of interest to note that a somewhat similar phenomenon to the fall in the attack-rate just discussed has been observed in Europe. In Nothnagel's Encyclopedia[5] we find the following sentence:—

"Gründler declares that the origin of the numerous pneumonic affections which he has noted in the garrison of Magdebourg from the 1st of October, 1873, to the end of June, 1874, was due less to the influence of the weather than to the arrival and education of recruits. . . . The complete strength of the garrison was 6,008 men, consisting of:—

Recruits, or men in 1st year of service: Pneumonia attack-rate ... ... 2·15%

Men in 2nd year of service: Pneumonia attack-rate 0·81%

Men in 3rd year of service: ,, ,, 0·67%

Men after 3rd year of service: ,, ,, 0·14%."

Aufrecht, the author of the article, considers that this predisposition of recruits is due to, "either unaccustomed or excessive muscular exertion, which may give rise to an enfeeblement of the entire muscular system, and, in connection with this, to an increased function of the sweat-glands." Further, after quoting some figures due to Riebe, he states:—"Any one who is not absolutely and wilfully blind cannot fail to see that the explanation of this state of affairs is to be found in the excessive demand on the powers of the young recruit and in the unwonted bodily exertion incident to the first year of service." These figures are not given in sufficient detail to enable us to judge whether they would if further sub-divided correspond in type to the distributions obtained from our Native returns; but if they did so correspond it would surely be only the "wilfully blind" who would be content to accept "either unaccustomed or excessive muscular exertion" as a sufficient explanation. In the case of the Natives referred to in Table III., excessive muscular exercise cannot explain the high attack-rate, nor could it account for the rate during the first month as given in Table IV. It has already been pointed out that all "Tropical Natives" have had, prior to their arrival on the Rand, a voyage of some ten days, followed by a detention in Ressano Garcia; and it is during their stay in the W.N.L.A. Compound in Johannesburg that the highest attack-rate from pneumonia occurs, and, therefore, before they commence the hard work incidental to mining. If, however, we had dealt with our data only in yearly groups, as in Gründler's returns, Aufrecht's suggested explanation might in this case also have appeared valid.

These figures of Gründler's are also quoted by Musser & Norris,[6] in support of a plea that occupation "plays an etiological rôle," and these authors further state "emigrants (*sic*) are more susceptible than Natives who have become acclimated." If this remark may be interpreted as meaning that immigrants suffer more from pneumonia than a native population of the same age and sex, it would have an important bearing on this problem. As an example of the contention that change of residence tends to increase the risk of contracting pneumonia they quote the case of the Lapps, but without giving their authorities or reproducing any figures, thus:—

"This is well exemplified in the case of the Lapps, who whenever they move from the frozen north to a temperate zone almost invariably die of pneumonia within a short time." On the other hand, Sir W. Osler[7] writes as follows:—"Newcomers and immigrants are stated to be less susceptible than native inhabitants." This view is directly opposed to those given above, but in the absence of the data on which these views are based it is impossible to judge of their value.

The influence of season and climate must now be considered in so far as the available data permit. We have seen that for the territorial groups the attack-rate and death-rate are highly correlated, and we find from the figures in Table V., page 16, that this is also true for the monthly groups of this table.

The partial correlation for recoveries and deaths for constant population grouped according to months is:—

$${}_{p}r_{rd} = \cdot 8739 \pm \cdot 0336.$$

Thus again we see that there must be a high correlation between cases and deaths; or in other words, the death-rate will serve as a close guide to the attack-rate. The problem, whether the case mortality is dependent on the rate of attack will be discussed later; but as will be shown, there appears to be little or no association between variations in the case mortality and in the prevalence of the disease.

In Chart IV. the death-rate for each month from August to the following April, both months inclusive, is shown for three successive recruiting seasons. The deaths have been recorded against the month in which the attacks commenced. It will be noted that in the season 1911 to 1912 the rate was highest in the colder months, and fell very noticeably in December and January, thus agreeing with an opinion quite commonly held that pneumonia is more prevalent during the colder months. In the season 1910 to 1911 the rate was highest in March and April, but no marked variations occurred from August to February. In the season 1912 to 1913 the maximum rates occurred during the warm weather, a reversal of the condition so marked in the season of 1911 to 1912. If we assume that other predisposing factors remained approximately the same, then from this evidence it would be difficult to state that season had any constant influence on the prevalence of the disease, although the tendency of the figures as a whole suggests that the prevalence decreases in the warmer months. This is shown graphically by the curve which represents the average rates for the three years.

We have no right, except hypothetically for the purpose of a first approximation, to assume that other factors have remained unaltered. For instance, we know that owing to a dry season in Tropical Africa in 1911 to 1912 there was a consequent shortage of crops, and it is possible that this factor may have had an influence on the Natives

TABLE V.

MONTH.	Average monthly number of recruits in Compd.	PNEUMONIA.					OTHER DISEASES.	
		Cases.	Rate per thousand.	Died.	Rate per thousand.	Case Mortality per cent.	Died.	Rate per thousand.
1910.								
August ...	1,293	43	33·3	11	8·5	25·6	32	24·7
September ..	1,864	69	37·0	27	14·5	39·2	98	52·6
October ...	2,077	61	29·4	22	10·6	36·1	78	37·6
November ...	2,141	73	34·1	36	16·8	49·3	36	16·8
December ...	2,375	68	28·6	28	11·8	41·2	18	7·6
1911.								
January ...	1,829	41	22·4	19	10·4	46·4	22	12·0
February ...	1,827	47	25·7	22	12·0	46·7	31	17·0
March ...	2,200	135	61·4	54	24·5	40·0	53	24·1
April ...	1,722	94	54·6	33	19·2	35·1	1	0·6
May	343	3	8·7	0				
June	69	0						
July	2	0						
August ...	939	143	152·3	48	51·1	33·6	48	51·1
September	2,040	219	107·4	72	35·3	32·8	16	7·8
October .	1,609	143	88·9	43	26·7	30·0	6	3·7
November	816	30	36·8	9	11·0	30·0	16	19·6
December ..	1,085	26	24·0	6	5·5	23·0	8	7·4
1912.								
January ...	936	16	17·1	5	5·3	31·0	19	20·3
February .	1,669	39	23·4	17	10·2	43·6	20	12·0
March ...	1,993	84	42·1	34	17·1	43·8	74	37·1
April	1,749	85	48·6	47	26·9	55·4	19	10·9
May	258	1	3·9	1	3·9	100·0		
June	20	0						
July	4	0						
August ...	1,552	96	61·9	39	25·1	40·6	41	26·4
September . .	2,251	81	36·0	35	15·5	43·2	2	0·9
October ...	1,448	40	27·6	25	17·3	62·6	32	22·1
November ...	1,751	73	41·7	35	20·0	48·0	11	6·3
December	1,718	99	57·6	41	23·9	41·5	59	34·3
1913.								
January ...	2,484			62	25·0		73	29·4
February ...	2,290			49	21·4		28	12·2
March ..	2,226			26	11·7		17	7·6
April	1,170			11	9·4			

arriving in the season of 1912 to 1913; again, a large proportion of Natives who arrived between November, 1911, and November, 1912, were receiving prophylactic inoculations of pneumococcal vaccine.

Some additional information on this question of seasonal prevalence can be obtained by a consideration of certain returns applying to Natives at work on the mines. Through the kindness of Dr. G. A. Turner, I have been able to obtain figures giving the average monthly number of Natives employed on the mines, with the corresponding deaths from pneumonia, for five years; subdivided according to the territorial areas from which they were recruited. In Chart IV. the death-rates per month for a five-yearly average are shown. In Chart V. the death-rate occurring in each month has been plotted in percentages of the total rate. Four distributions obtained from data relating to European populations in different parts of the world are also shown for comparison. The two distributions obtained from figures relating to populations in the Northern Hemisphere have been plotted by reading July for January, and so on, so that the seasons shall correspond.

It will be noticed that the monthly fluctuations in the Native distributions are not so marked as those in the European distributions, and that Natives from British territory show a greater seasonal variation than those from Portuguese territory. Nevertheless, all groups show lower rates during the warmer months. In regard to British Central African; Mozambique; and Zambezi, Quilimane, and Tete Natives; it must be remembered that no new arrivals are being allotted to the mines from May to nearly the end of August, and this factor in itself will, as we have seen, tend to reduce the death-rate observed in these months. Monthly fluctuations among "Tropical Natives" are, however, much more marked; this is partly accounted for by the fact that the rates are calculated from smaller numbers.

Summing the whole of this evidence, we may, I think, conclude that pneumonia tends to be definitely more prevalent during the colder months. Fluctuations due to other causes, more especially amongst the "Tropical Natives," may, however, to a large extent mask the effect of season when small groups are dealt with. Further, the effects of seasons are probably dependent on the meteorological conditions with which they are associated; for instance, the temperature or rainfall of the May of one year might correspond more closely to that of the June than the May of a preceding, or following year. The agreement, therefore, between the rates and changes in meteorological conditions might be greater than that observed in the monthly charts. Alterations of weather conditions are, no doubt, highly correlated with season; these facts, nevertheless, must be borne in mind when attempting to interpret the meaning of the foregoing data.* Cold or a fall in temperature is the constant factor of the winter season in all parts of the world. In the interior of South Africa the winter season is the dry and dusty season, but in Northern Europe it is the damp season.

It is interesting to recall that this view of the influence of season on the incidence of pneumonia was realised by the Greek physicians. Two of the aphorisms of Hippocrates show this. Sec. III., No. 19: "All diseases occur at all seasons of the year, but certain of them are more apt to occur and be exacerbated at certain seasons." In the following aphorisms he proceeds to deal with those diseases prevalent in Spring, Summer, and Autumn, and No. 23 reads as follows: "Of Winter, pleurisy, *pneumonia*, coryza, hoarseness, cough, pain of the chest, pains of the ribs and loins, headache, vertigo, and apoplexy."[9]

To return to a consideration of the relationship of the attack-rate to the case mortality; this is of some importance from a statistical point of view, as well as, in its physiological aspect. It must be studied first of all in regard to the tribal distribution, and then in respect to season. The problem may be stated in this way; is a "tribe" that is especially prone to pneumonia liable to a higher case mortality than a "tribe" less liable to attack, or the reverse? In other words, does an increased liability to attack imply an increased liability to death when attacked? If this be so, then by using death-rates to judge of the prevalence of attack we should be exaggerating any differences in the attack-rates that may be present. The co-efficient of correlation for attack-rate and case mortality, in the case of the territorial distribution, is:—

$$r = \cdot 3895 \pm \cdot 1809$$

but this value is unfortunately not a safe guide owing to mathematical difficulties, and to the introduction of a spurious correlation due to the peculiar nature of the problem. If r = recoveries and d = deaths, the case mortality as usually expressed $= 100 \dfrac{d}{r + d}$. If the case mortality is constant, then $\dfrac{d}{r + d}$ is constant, and d is proportional to r. If $\dfrac{d}{r+d} = x$ then $d = r \dfrac{x}{1 - x}$. The partial correlation, therefore, of r with

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* It will be noted that the seasonal prevalence of pneumonia which is similar in the figures obtained from Europe, America, Australia, and South Africa, markedly diminishes with the advent of warmer weather, and increases again as the winter approaches. This phenomenon is not consonant with the theory which has recently been suggested by some, that the house fly is an important factor in the dissemination of this disease.

d for x constant will be unity. In so far as the value found falls short of unity it will be a measure of the variability of x, i.e., the case mortality.

We have already found the partial correlation:—

$$_pr_{rd} = \cdot 9045 \pm \cdot 0388$$

and this when considered with its probable error leaves very small grounds for any assertion that the case mortality varies, to any appreciable extent, with the attack-rate.

Turning now to seasonal data, we find, correlating directly the attack-rate with case mortality:—

$$r = -\cdot 2143 \pm \cdot 1342.$$

The partial correlation of recoveries with deaths for constant population has been shown to be:—

$$_pr_{rd} = \cdot 8739 \pm \cdot 0336.$$

Here, then, it would seem that there is rather more justification for assuming that the case mortality is not, within the limits of random sampling, a constant.

The crude correlation for the rates in these two instances gives values which, when considered in relation to their probable errors, cannot be regarded as significant, and are of unlike sign.

It would appear, therefore, that the case mortality certainly does not bear any close relationship to the attack-rate; and the figures before us suggest that an increase in the attack-rate is not associated with an increase in the case mortality from the disease.

SECTION II.

The Clinical Manifestations, Course, Symptoms, and Gross Morbid Anatomy of Pneumonia.

The clinical signs and symptoms of an uncomplicated case of pneumonia in a "Tropical Native" do not differ materially from those in a European. There is, however, a marked difference in temperament; the Native being apparently much less sensitive to the discomfort of illness than the European. As a result he frequently remains at work when seriously sick unless under careful European supervision. Although the onset of pneumonia is usually sudden and accompanied by high fever, yet not infrequently the Native will fail to report sick if left to himself.

Out of 975 cases which recovered, the fever terminated by lysis on 503 occasions (51·6 per cent.); by crisis on 338 occasions (34·7 per cent.); and its termination was atypical—i.e., could not satisfactorily be classified under either of the previous headings—in 134 cases (13·7 per cent.). In the European statistics which I have been able to consult the manner in which the fever terminated is usually classed into only the two groups, crisis or lysis; and no doubt some of those cases which are here grouped as atypical would appear in the lysis group, while others would possibly have been classed as crisis. The term "crisis" as used in this classification is understood to denote a sudden and continued fall of temperature to the normal, which is reached within about thirty-six to forty-eight hours of the commencement of the fall. Where a sudden fall has been followed by an intermittent temperature for several days, as is not uncommon, the case has been classed as atypical, as also cases in which the temperature has been intermittent from the commencement, apyrexial cases, and so on. Among the 134 cases classed as atypical 23 were afebrile, and four were not admitted to hospital until after the temperature had fallen to normal.

To consider first those cases which terminated by *crisis*. Out of a total of 378 cases there were 43, or 11·4 per cent., that died after the temperature became normal; 25 of these were, so far as a macroscopic post mortem examination could determine, unaccompanied by any pathological process other than a pneumonic consolidation in the lungs. Twelve of these deaths occurred within 24 hours of the time the temperature reached the normal; the remainder were distributed as follows:—One on the third, three on the fourth, fifth and seventh respectively, two on the ninth, and one on the seventeenth day.

Chart VI. shows the distribution of cases according to the day after admission on which the temperature reached the normal, which, for practical purposes, we may term the *day of crisis*. For comparison a similar distribution of 4,718 cases recorded by Musser & Norris is shown, and also a series of 873 cases by Aufrecht.[5] The chief constants of these distributions are given in the following table:—

TABLE VI.

	"Tropical Natives."	Musser & Norris	Aufrecht.
Mean ...	5·3519 ± ·0849	6·6045 ± ·0234	6·7211 ± ·0596
Mode ...	4·5733	5·8797	5·9871
σ ...	2·4483 ± ·0600	2·3450 ± ·0165	2·6088 ± ·0421
β_1 ...	0·2968 ± ·0954	0·5725 ± ·0661	0·5684 ± ·1833
β_2 ...	3·2219 ± ·2657	4·2656 ± ·2622	4·4898 ± ·8972
Type ...	I.	IV.	IV.
Number ...	378	4718	873

Here a word of caution is necessary, a death occurring in the first twenty-four hours is considered as occurring on the first day, a death occurring in the middle of the sixth day would correspond to a position on the chart of 5·5 on the abscissa; thus the mean day of crisis is given as 5·35 days, that is, it falls in the sixth day. A reference to Chart VI. will show that the two European distributions are similar in type and position; and considering the value of their constants in relation to the probable errors their similarity suggests that these curves closely represent the usual distribution for "crisis" in pneumonia amongst Europeans in the Northern Hemisphere. Both distributions are definitely leptokurtic, while that obtained from the Native data is, within the limits of errors of sampling, mesokurtic. The standard deviation of all three distributions is approximately the same. The most marked difference between the Native and European curves is, that the former may be described as having been bodily moved to the left by about a day and a half; this, if it be significant of a difference in the type of disease, would mean that the course of the illness is, on the average, a day and a half shorter. It will no doubt occur to the reader that this difference is possibly due to Native admissions to hospital not taking place until about thirty-six hours after the commencement of the illness, but I do not think this is the probable explanation. Although a Native is often slow to voluntarily report himself sick, yet in the W.N.L.A. Compound the police boys inspect all compound rooms several times a day in order to discover and report any " boy " who appears sick; and, as the onset of this disease is usually more or less sudden and severe, it seems hardly likely that we must allow an average of thirty-six hours between the onset of symptoms and admission to hospital. Further, the greatest number of admissions takes place within the first twenty-four hours of the arrival of the Natives on the Rand. During the journey from Ressano Garcia they are under the supervision of a competent European, who sends any sick " boy " to hospital immediately on arrival. If it should be thought that the large number of admissions occurring immediately after the arrival of the Natives on the Rand be due to an accumulation of cases that developed the disease prior to their arrival, then the case mortality and length of illness after admission to hospital might be expected to throw some light on such a proposal. These problems are considered on pp. 25 and 34, and it will be seen that there is no evidence to support such a suggestion.

If we can dismiss as unfounded the supposition that the alteration in the position of the Native curve be due to the delayed admission of cases to hospital, then we have evidence that pneumonia runs a shorter course amongst " Tropical Natives " than amongst Europeans in the Northern Hemisphere.

Turning now to an examination of the cases classed under the heading *lysis*. The distribution of these cases, grouped according to the day on which the temperature reached the normal, is shown in Chart VII. It is more difficult to obtain data from European sources for comparison. Aufrecht in Nothnagel's Encyclopedia[5] writes:—" It is well in all cases in which the drop in temperature occurs by lysis to look for the existence of a complication. Even if it is not proper in such cases to exclude a true pneumonia and accept pneumonic relapses as the most frequent cause, nevertheless, complications may be the cause of a fall in temperature by lysis." The only figures he quotes are the following from Wagner:—" In the years 1881 to 1883 he (Wagner) saw among 199 pneumonias only 115 termniate by crisis (26 by protracted crisis), and 84 by lysis." That is 42 per cent. of these cases ended by " lysis," and 45 per cent. by what would in this paper have probably been called " crisis." Of the 975 cases in our records 52 per cent. terminated by " lysis," and 35 per cent. by " crisis." I have been unable to find any figures from European cases which enable a detailed comparison with the Native cases to be made. Reference to the chart will show that the commonest day for the temperature to reach normal by " lysis " is approximately two days later than that for " crisis "; that is, the mode is at 6·5 days as against 4·3 in the " crisis " distribution.

The constants of the "lysis" distribution are given in the table below:—

TABLE VII.

Constants of distribution when termination is by Lysis.

Mean	$7 \cdot 4609 \pm 0 \cdot 0802$
Mode	$6 \cdot 5028$
σ	$2 \cdot 8234 \pm 0 \cdot 0567$
β_1	$0 \cdot 2924 \pm 0 \cdot 0724$
β_2	$3 \cdot 1272 \pm 0 \cdot 1982$
Type	I.
Number	564

Both the "crisis" and the "lysis" distributions are very closely similar in regard to their variability, skewness, and kurtosis, the difference in the positions of the mean and mode is, however, marked. This shifting of the mode may be a measure of the extra time required for the temperature to fall to normal, and suggests that if we had been able to date the records of the cases from the commencement of the fall in temperature the two distributions might have been almost identical. If this be so, the explanation of the cause of a fall of temperature by "lysis" as suggested in the extract quoted above would not appear to be satisfactory, at any rate, if applied to the pneumonia of "Tropical Natives."

There are, however, appreciable differences between the types of the disease as met with among these Natives and the European examples quoted; first, in the duration of the febrile period, and secondly, in the smaller percentage of all cases which terminate by "crisis."

If we form a table by entering each case according to day of termination by crisis, and day of admission to hospital after arrival in compound, we find that the coefficient of correlation is zero, for

$$r = -\cdot 0035 \pm \cdot 0346,$$

and thus we may conclude that Natives attacked immediately on arrival are neither more nor less likely to have a prolonged illness than those attacked later. This value also indicates that attacks do not commonly commence before arrival in the compound, for if they did, we might reasonably expect that the length of the febrile stage in hospital would be of shorter duration; unless, indeed, we assume that the exposure and lack of proper treatment before admission increased the severity of the attack, in which case the mortality among the earlier cases should be higher than the later, which it is not.

The same statements apply to the group of cases in which the pyrexial stage terminates by "lysis." From a similarly constructed table the value of the correlation is found to be

$$r = -\cdot 0258 \pm \cdot 0282.$$

The ratio of cases in which the fever terminated by "lysis" to those terminated by "crisis" is shown in Table VIII., for periods of five days.

TABLE VIII.

Ratio of cases terminating by lysis to those by crisis.

Days	...	Total	0-4	5-9	10-14	15-19	20-24	25-29	30 and over.
Crisis	...	378	122	81	59	46	33	7	30
Lysis	...	564	167	127	99	95	37	15	24
Ratio	...	149	137	157	168	203	112	214	80

We have already referred to the hypothesis that a termination by "lysis" indicates a different type of attack, and have quoted one author's opinion that a fall of the temperature by "lysis" probably indicates the presence of some complication; we can, therefore, proceed to examine this problem from another point of view. The duration of convalescence after the temperature has reached the normal will enable us to form some opinion as to the degree of severity of the attack, and also as to the probable presence of complications. Table IX. gives the chief constants of the distributions for recoveries from pneumonia after termination of the fever by "crisis" and "lysis" respectively, according to the number of days spent in hospital after the temperature reached normal. Cases in which a second attack followed the first before discharge from hospital have been omitted from both distributions.

TABLE IX.

Constants of distribution for days in hospital after the febrile stage.

		Crisis.	Lysis.
Mean	...	10·9697 ± 0·1906	10·2889 ± 0·1589
Mode	...	5·3337	5·4325
σ	...	5·1336 ± 0·1348	4·9858 ± 0·1124
β_1	...	1·8809	2·0976
β_2	...	5·0645	5·5951
Start	...	5·1036	4·8854
Number	...	330	488
		Type I.	Type I.

There is no significant difference between these two distributions, and any suggestion that a fall of temperature by "lysis" is due to complications which are likely to delay convalescence is, as regards these figures, quite unjustified. Further, we may remark that there is only a very small correlation between the day of illness on which "crisis" took place and the length of time spent in hospital after the temperature became normal; that is, patients who have a short attack of fever do not seem on the average to convalesce more rapidly than those in which the pyrexial stage lasts longer. It may here be remarked, that usually owing to pressure of cases for admission the patients are discharged from hospital as soon as it is safe to do so. In the W.N.L.A. Hospital there is no routine period of retention after the febrile stage is over. The absence of any fixed period of retention is shown by the variability of the distributions—as shown in Table IX. by the standard deviations. The pressure of new cases sometimes necessitates the discharge of cases to the convalescent rooms before they would otherwise have been so transferred, and it is probable that those whose attack is of shorter duration would, other things being equal, be the first to be sent out, and thus this factor may possibly account for the small correlation actually found. The values are:—

(1) Day on which temperature reached normal by "crisis" with days spent in hospital after normal temperature was reached.

$$r = \cdot 0823 \pm \cdot 0369.$$

(2) Day on which temperature reached normal by "lysis" with days spent in hospital after normal temperature was reached.

$$r = \cdot 1194 \pm \cdot 0301.$$

It would seem then that the fall of temperature to normal marks a definite stage in the disease, probably the termination of the infective process, and that thereafter the stage of convalescence is very little influenced by the length of the previous pyrexia.

It is of some importance to try and determine on rational grounds the stage at which we may consider the disease to have ended, in order that we may be able to distinguish between an extension of an established pneumonic process and a new attack.

In considering the question of the termination of the attack in the light of modern theories of acquired immunity, the investigation of second attacks is of considerable interest and importance. We must first consider what meaning we shall attach to the terms "second attack," "recurrence," and "extension of the disease." Is there any essential difference in meaning between these terms?—and if so, in what does it consist? It is quite easy to understand that a gradually spreading pneumonic process in a lung, where we may assume that immunity has not yet been established, is totally different to a new infection occurring after a period of return to normal health; but is there any essential difference in the pathological process involved in a gradually spreading consolidation, and a more or less sudden affection of a hitherto unaffected lobe during the course of the febrile period? If we assume that these two conditions are but different manifestations of a similar process then we must enquire at what stage in the course of the disease, or at what stage during or after convalescence, we are entitled to consider the reappearance of symptoms as a "second attack." It will be noticed that the word "recurrence" is not used, because this word does not seem to denote a process essentially different from a second attack, unless it refers to a recrudescence of the original infection. In this case, the time factor alone can separate it from "extension of the disease," and any such difference must of necessity be, it seems to me, purely arbitrary. The term "recurrence" seems to be applied indifferently to two probably separate conditions, and in this category may be found cases belonging to both the above classes; if this be so, then the use of this word merely serves further to obscure an already somewhat difficult problem. It will, I think, be readily admitted that if the length of time after the first invasion be the only difference between a "recurrence" and a "second attack" it will help to simplify our ideas if we drop the former term; if on the other hand an essential difference between the processes exist we must reintroduce it when the difference has been scientifically defined.

In this connection the leading text-books of medicine do not, unfortunately, give us much assistance. Sir W. Osler,[7] under the heading *Previous Attack*, writes:—"No other acute disease recurs in the same individual with such frequency. Instances are on record of individuals who have had ten or more attacks. The percentage of recurrences has been placed as high as fifty. Netter gives it as thirty-one, and he has collected the statistics of eleven observers who place the percentage at 26·8. Amongst the highest figures for recurrences are those of Benjamin Rush, 28, and Andeal, 16." Under the heading *Relapse*, we find the following remarks:—"There are cases in which from the ninth to the eleventh day the fever subsides, and after the temperature has been normal for a day or two a rise occurs and fever may persist for another ten days or even two weeks. Though this might be termed a relapse, it is more correct to regard it as an instance of an anomalous course of delayed resolving. Wagner, who has studied the subject carefully, says that in his large experience of 1,100 cases he met with only three doubtful cases. When it does occur, the attack is usually abortive and mild." The meaning of this quotation appears to me obscure. In the first extract "previous attacks" are spoken of as "recurrences," and we must therefore regard these terms as synonymous; further we are told that such events occur frequently. A "relapse" is apparently some condition of rare occurrence, and the context makes it impossible to give the word its usual meaning, as applied to the patient in contradistinction to a phase of the disease.

Aufrecht, in the article "Inflammation of the Lungs,"[5] writes:—"Also in adults pneumonias may occur repeatedly. . . . According to Riesell's experiences, among every hundred pneumonias almost regularly 50 represent first attacks, 32 second, 15 third, while the remaining 3 represent a fourth, or even more frequently repeated attack." Musser & Norris[6] state:—"Among the writer's cases collected in reference to this subject 1,615 out of 11,811 had previous attacks (13·6 per cent.), and of 246, 189 were in the second, 33 in the third, 18 in the fourth, 3 in the fifth, 1 in the sixth, 1 in the eighth, and 1 in the ninth attack."

These extracts show that "recurrences," and "second attacks" are well recognised conditions, and not uncommon; but no definition of the differences, if any be implied by these terms, is given

In any case of pneumonia the consolidation may be limited to one lobe, but not infrequently two or more lobes are affected. If the neighbouring lobe is most commonly involved by a direct extension of the infective process we ought to find two adjacent lobes more often attacked than two lobes not in direct contact. A reference to Table X., page 28, shows that there were 247 cases in which two lobes only were affected, and gives the distribution of the pairs affected. The data in this table were obtained entirely from post mortem examinations. Out of 213 cases where only one lobe was affected the percentage distribution was as shown in Column (1). Assuming that the first lobe of the affected pairs followed a similar distribution, and that of the remaining four unattacked lobes the rate of those attacked would be in a similar ratio, we can calculate the distribution of pairs to be expected. It is given in Column (2) to the nearest unit. The observed distribution is shown in Column (3), and the differences in Column (4).

We see that adjacent lobes, that is the upper and middle, and middle and lower, on the right side, and the upper and lower on the left, are more frequently both attacked than they ought to be on our assumption, while one lobe of each lung, excluding both lower lobes, occurs much too seldom. It may quite fairly be objected, that considering the anatomical structure of the right lung, we are not justified in regarding the middle lobe as an anatomical entity, and that it should be considered merely as a portion of the upper lobe; for much of it is directly continuous with this lobe. If then we recalculate our table and add to the total number of pairs formed by the upper and lower, and middle and lower, the group "all three right lobes" we obtain by a similar process the results shown in Table XI., page 28.

TABLE X.

		Distribution per cent. of lobes affected when only one lobe is involved. (1)		Expected Distribution for pairs. (2)	Observed Distribution for pairs. (3)	Difference (Column 3 minus Column 2.) (4)
R.	Upper ...	24·0	Upper & Middle	4	16	+ 12
	Middle	2·8	Lower ,, ,,	5	16	+ 11
	Lower ...	30·4	R. Upper & Lower	50	37	— 13
L.	Upper ...	6·7	L. Upper & Lower	16	68	+ 52
	Lower ...	36·1	Both Upper	9	6	— 3
			,, Lower	82	83	+ 1
			Unpaired lobes in two lungs	81	21	— 60
			Total ...	247	247	

TABLE XI.

	Expected distribution for pairs.	Observed distribution for pairs.	Difference.
R. Upper and Lower	85	151	+66
L. Upper and Lower	17	68	+51
Both Upper ...	16	6	–10
Both Lower ...	92	83	– 9
Unpaired lobes in two lungs ...	119	21	–98

TABLE XII.

	Expected distribution for pairs.	Observed distribution for pairs.	Difference.
R. Upper and Lower	86	151	+65
L. Upper and Lower	19	68	+49
Both Upper ...	18	6	–12
Both Lower ...	90	83	– 7
Unpaired lobes in two lungs ...	116	21	–95

Again we see that an affection of adjacent lobes is in very marked excess of the theoretical number. From both tables we may conclude —accepting the above hypothesis as correct—that at least one-third of the infections of second lobes are due to direct extension of the disease from lobe to lobe. If we further assume that the reverse of the proposition is also true, and that in two-thirds of the cases the second lobe becomes affected by a new focus of the disease (arising from some cause other than direct extension), we must also assume that this new centre might appear in the lobe which had been first affected. Reconstructing our table on this assumption, we obtain the results shown in Table XII., page 28.

This again demonstrates the same phenomenon, and we have, therefore, some justification for assuming that the infective process may spread by direct extension from lobe to lobe.

Let us provisionally consider all cases in which the temperature rises after it has been normal for twenty-four hours or more, when the rise is accompanied by a reappearance of the signs and symptoms of pneumonia, to be "second attacks." The cases have been divided, (Tables XIII. and XIV.) according to the length of the period between the end of the first attack, as previously defined, and the commencement of the second; so that if this definition does not satisfy the reader he can see how great, or how little, a difference will be made by excluding such cases as he may desire from the distribution, and considering them as recrudescences of the first attack.

When attempting to estimate the frequency of second attacks considerable difficulty arises in determining the size of the population in which the second attacks arose, and only in the "8,000 experiment" can this be obtained with approximate accuracy for a period exceeding one month. From the number attacked during each month of residence on the Rand must be deducted (1) the number of deaths during first attacks, (2) the number of deaths from other causes, and (3) the number repatriated. The resulting populations are given in Table XIIIA. This table is to be read thus: In the first row, and first column, is found the number 168, which indicates that there were 168 Natives who recovered from an attack of pneumonia contracted during their first month of residence on the Rand, and who remained under observation for thirty days after recovery. In the first row, and second column, the figure 151 occurs, which indicates that the population from which second attacks were possible during the second period of thirty days from the end of a first attack contracted within a month of arrival, had been reduced by seventeen. In Table XIIIB. the actual number of second attacks is given, according to the date of occurrence of first attack, and the length of time after recovery before the second was contracted. In Table XIIIC. the rates per month per thousand of population are given.

TABLE XIII. A.

Population, i.e., number of "boys" exposed to risk of second attack.

Month after arrival in which 1st attack occurred.	Months after termination of 1st attack.						
	1	2	3	4	5	6	Total.
1st	168	151	139	127	115	104	804
2nd	136	133	123	115	113		620
3rd	92	85	80	75			332
4th	53	48	47				148
5th	76	73					149
6th	40						40
Total	565	490	389	317	228	104	2093

TABLE XIII. B.

Number of second attacks.

Month after arrival in which 1st attack occurred.	Months after termination of 1st attack.						
	1	2	3	4	5	6	Total.
1st	12	2	6	3	5	4	32
2nd	1	5	4	1	0		11
3rd	5	2	3	0			10
4th	2	0	0				2
5th	2	2					4
6th	1						1
Total	23	11	13	4	5	4	60

TABLE XIII. C.

Rates per thousand.

Month after arrival in which 1st attack occurred.	Months after termination of 1st attack						Average Monthly Rate.
	1	2	3	4	5	6	
1st	71	13	43	24	43	38	40
2nd	7	38	33	9	0		18
3rd	54	24	38	0			30
4th	40	0	0				14
5th	26	27					27
6th	25						25
	41	22	33	13	22	38	28·7

It will be noticed that there is apparently a tendency for the rates to fall as the length of residence on the Rand increases, a phenomenon also observed for first attacks. Further, although the rates are irregular owing to their having been calculated from small numbers, there seems to be a tendency for second attacks to become less common as the interval after the first increases. It must be remembered that the numbers in column (1) of Table B. must be too small, as second attacks, which occur before discharge to work, were not notified by the mine authorities; and there will, therefore, be few or no cases in the mine records occurring within fourteen to twenty-one days after the first attack. The mean monthly attack-rate for the six-month period is 28·7 per thousand, which, as we have just seen, must be somewhat too low for the period dealt with in the tables.[†] The mean monthly attack-rate for first attacks out of the whole population for the same period is 22·3 per thousand.

Out of 1,129 Natives who recovered from pneumonia in the W.N.L.A. Compound Hospital eighty had a "second attack" within thirty days from the termination of the pyrexial period of the first attack; the distribution in days after first attack is shown below:—

TABLE XIV.

Day after termination of fever of first attack.	Number of cases of "second attacks."	
0	1	30
1	4	
2	12	
3	4	
4	5	
5	4	
6—10		17
11—15		14
16—20		12
21—30		7
Total		80

The population throughout this period remained practically constant, and we therefore find a rate of seventy per thousand for second attacks occurring within thirty days of the end of the first attack, as against a rate of forty per thousand for primary pneumonias

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[‡] Owing to the construction of the table, the rates for the columns will be progressively too high as the period increases; for the attack-rate for second attacks apparently decreases with increase both in period of residence and length of interval after the first attack. This defect in the table is unavoidable, as the period of observation was limited to six months. Further, in reference to this table the remarks made in connection with the effect of prophylactic inoculation must be borne in mind (see p. 89).

originating within about a month from the date of arrival of the Natives in Johannesburg.

These figures, showing that amongst "Tropical Natives" in the W.N.L.A. Compound "second attacks" amongst the attacked are more frequent than "first attacks" among the whole population, may indicate that one attack predisposes to a second; or that we are dealing with a particularly susceptible population selected as such by the first attacks. The question is one of considerable difficulty, and in considering it one must not lose sight of the fact that "second attacks" appear to become less common as the period after the first increases; if this be a rule then it would lend support to the suggestion that the first attack actually predisposes to the second. The mechanism of recovery would, however, be difficult to understand on the basis of modern theories of immunity, unless we may accept the suggestion, put forward in the footnote on p. 42, that real differences in strains of pneumococci do occur, and are of pathological significance.

The distribution given in Table XIV. is shown graphically in Chart IX. with its theoretical curve. This curve is of the "J" type, and starts at 0·45 of a day after the termniation of the pyrexial period of the first attack and ends at 36·03 days. It is well known that the probable error of the start and end of these "J" shaped curves is large, and further, that no satisfactory correction for the crude moments has as yet been described; we must not, therefore, lay too much stress on the fact that the theoretical curve terminates as shown. This fact, however, may suggest that second attacks are not all similar in character, and that those occurring after, say, the first month, owe their origin to a different set of causes. Moreover, there is in our previous figures (Table XIII.) a suggestion that the rate may fall in the second month after attack and rise again in the third, but owing to the smallness of the numbers and to other sources of error it will not be wise to do more than note these irregularities. Thus while not affirming that there is any demonstrable difference in the earlier and later distributions of second attacks, we must keep an open mind on the question until fuller data are available for investigation.*

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* The medical officers of the Crown Mines have very kindly obtained for me the subsequent history of all cases of pneumonia occurring amongst *all* Natives employed on these mines for a period of one year, and the number of second attacks. These returns enable us to calculate the population at risk for each month for a twelve months table; but only tables corresponding to those of Table XIII. are here reproduced. Two cases of second attacks occurred in the portion of the table not reproduced, and the monthly attack-rate for second attacks for the twelve months table is 7·5 per thousand. It should not be overlooked that these figures refer to all Natives—Union Natives, East Coast Natives, and "Tropical Natives," and are not confined to the latter group as are those of Table XIII. The

primary attack-rate for pneumonia is much lower among the former groups than among "Tropical Natives." The total monthly attack-rate for the year 1912-1913 was approximately 3·80 per thousand.

Tables for "second attacks" from Crown Mines data:—

A. Population, i.e., number of "boys" exposed to risk of second attack.

Month after arrival in which 1st attack occurred.	Months after termination of 1st attack.						
	1	2	3	4	5	6	
1st	106	95	85	75	65	47	473
2nd	93	83	74	68	61		379
3rd	55	47	43	36			181
4th	52	46	40				138
5th	35	32					67
6th	31						31
	372	303	242	179	126	47	1269

B. Number of second attacks.

Month after arrival in which 1st attack occurred.	Months after termination of 1st attack.						
	1	2	3	4	5	6	
1st	0	1	1	0	0	0	2
2nd	2	1	0	1	2		6
3rd	1	0	1	0			2
4th	0	2	2				4
5th	0	0					0
6th	0						0
	3	4	4	1	2	0	14

C. Rates per thousand.

Month after arrival in which 1st attack occurred.	Months after termination of 1st attack.						
	1	2	3	4	5	6	
1st	0	11	12	0	0	0	4
2nd	22	12	0	15	33		16
3rd	18	0	23	0			11
4th	0	44	50				29
5th	0	0					0
6th	0						0
	8	13	17	6	17	0	11

Reference has already been made to the case mortality among primary attacks, and we have seen (Table II.) that the average rate for all groups of "Tropical Natives" attacked in the W.N.L.A. Compound is 39·2 per cent. The European case mortality for this disease is given in the text books as varying from 5 to 50 per cent. and over, for persons between 20 to 40 years of age. The European hospital case mortality is recorded as being from 20 to 40 per cent., but is on the average, higher than that obtained from the returns of private practitioners. Whether this difference is a result of hospital treatment, or is due to the selected character of the hospital population, it is impossible to judge, as I have been unable to find any records of the case mortality for patients who might be fairly considered comparable to those usually found in hospital, but who have, notwithstanding, been treated outside. We have already seen that there is probably no appreciable correlation between variations in the attack-rate and the case mortality, whether the increased attack-rate be "tribal" or "seasonal" in origin. The case mortality does not show any reduction with an increase in the length of residence although the attack-rate rapidly falls. Table XV. gives the rates for five-day periods.

TABLE XV.

Days after arrival on the Rand before admission to hospital.

Day after arrival ...	0-4	5-9	10-14	15-19	20-24	25-29	30-34	35-	Total
Cases	525	388	314	275	157	55	37	58	1809
Deaths	186	155	140	100	59	31	13	25	709
Case mortality per cent.	35·9	39·8	44·6	36·4	37·6	56·4	35·1	43·2	39·2

We should not expect, therefore, to find that the case mortality from second attacks would be lower than that from first attacks on the ground that a second attack must occur at a later date.

Out of sixty cases of second attacks occurring over a period of six months in the group of 8,123 "boys" (the "8000 experiment") there were thirteen deaths, giving a case mortality of 21·7 per cent., as against a case mortality of 32·1 per cent. for first attacks in this group. Out of eighty second attacks occurring in the W.N.L.A. Compound Hospital twenty-six died, giving a case mortality of 32·5 per cent., as against 39·2 per cent. for first attacks occurring in the same group. Thus in both groups we see that there is an apparent reduction in the case mortality from second attacks; at any rate, when they occur within a comparatively short period after the first. It must be remembered that the numbers are small and the probable errors, therefore, large. Using the ordinary formula $m\bar{p} \pm \cdot 67449$

$\sqrt{m\bar{p}\bar{q}}$ (which considering the relative size of $\bar{p}$ and $\bar{q}$ is probably legitimate), we find the expected number of deaths in the first case to be $19{\cdot}5 \pm 2{\cdot}44$ as against the observed 13, and $31{\cdot}6 \pm 2{\cdot}94$ as against the observed 26 in the second. If we form a "four fold table," and calculate the value of χ^2 in each case we reach $P = {\cdot}4016$ and $P = {\cdot}7149$.* These values show that the results may not improbably have arisen owing to errors of small samples. This reduction in case mortality, if, however, it be significant, may perhaps be due to the fact that those individuals who are least able to resist the invasion are eliminated from the population by the first attack, and if this were so, any assumption in regard to an acquired resistance by virtue of a first attack would be unnecessary.

It may be pointed out that third, and even fourth, attacks may occur within short periods. Amongst eighty Natives who contracted second attacks and whose history is known for a period of six months, six had a third attack, and one of these a fourth.

In Table XVI., p. 36, an analysis of the macroscopic *post-mortem* appearances in 688 cases of pneumonia is given. The term pneumonia, here as elsewhere, is applied only to cases in which consolidation of the lung was present, and fatal cases of pneumococcal infections not accompanied by consolidation of the lung have been omitted, as for example pneumococcal meningitis unaccompanied by pneumonia.

Meningitis was a complicating factor in 10·5 per cent. of the fatal pneumonias recorded in this table. In 85 per cent. of these cases the vertex of the cerebrum was affected, and this accords with the experience of other observers that this site is commonly involved in meningitis of pneumococcal origin. The number of these cases of pneumonia complicated with meningitis gives a much higher rate than those recorded by Musser & Norris,[6] or by Aufrecht.[5] In our series the cranial cavity was examined in every instance, and we are not informed whether this precaution was always taken by the observers quoted by these authors. The insidious onset of this complication and the uncertain character of the symptoms often makes the diagnosis of pneumococcal meningitis during life one of great difficulty, and in practice it is not rare to find exudation on the meninges in cases in which meningitis had not been suspected during life. The absence of symptoms may be due to the fact that not infrequently the vertex is the only region affected. Musser & Norris write:—" Among 49,028 collected clinical cases meningitis occurred in 206 (0·24 per cent. (sic)), and at autopsy in 180 out of 4,833 (3·5 per cent. (sic)). The percentages are actually 0·42 per cent. and 3·7 per cent. respectively, if the numbers stated are correct. Aufrecht writes:—

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* See footnote to p. 52.

MORBID ANATOMY.

TABLE XVI.

Analysis of macroscopic leasions found on post-mortem examination of 688 fatal cases of pneumonia amongst "Tropical Natives" dying in the W.N.L.A. Compound Hospital on the Rand.

	Organ affected.	Number of Cases	Percentage in sub Group.	Percentage of Total Cases.	
Meningitis.	Vertex	25	34·7	3·63	
	Base	7	9·7	1·02	
	Ventricles	2	2·8	0·29	
	Vertex and Ventricles	2	2·8	0·29	
	Base and Ventricles	2	2·8	0·29	
	Base and Vertex	17	23·6	2·47	
	Base and Vent. and Vert.	17	23·6	2·47	
	Total	72	100·0		10·17
Peri-carditis.	Purulent	25	37·3	3·63	
	Serous	42	62·7	6·11*	
	Total	67	100·0		9·74
Recent Pleurisy.	Right Chest	92	33·1	13·37	
	Left Chest	63	22·7	9·16	
	Both Chests	123	44·2	17·88	
	Total	278	100·0		40·41
Empyema		19			2·76
Right Lung.	Upper Lobe	51	17·6	7·41	
	Middle Lobe	6	2·1	0·87	
	Lower Lobe	65	22·5	9·45	
	Upper and Middle	16	5·5	2·33	
	Middle and Lower	16	5·5	2·33	
	Upper and Lower	37	12·8	5·38	
	Three Lobes	98	33·9	14·24	
	Total	289	100·0		42·01
Left Lung.	Upper Lobe	14	8·8	2·03	
	Lower Lobe	77	48·4	11·19	
	Both Lobes	68	42·8	9·88	
	Total	159	100·0		23·11
Both Lungs.	Both Bases	83	39·5	12·06	
	Both Apices	6	2·9	0·87	
	Two Lobes	21	10·0	3·05	
	Three Lobes	54	25·7	7·85	
	Four Lobes	37	17·6	5·38	
	Five Lobes	9	4·3	1·31	
	Total	210	100·0		30·52
Lung not stated		30			4·36
	Total	688			100·0

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* This group may include some cases of serous transudation without pericardial inflammation.

"The frequent occurrence of meningitis in the course of pneumonia has been emphasised by Immermann & Hiller on the basis of earlier reports in literature. . . . . I myself in 253 cases, in almost all of which an autopsy was performed, found meningitis but seven times; besides this, I have noted meningitis three times as a complication of pneumonia in patients who recovered. According to this, among my 1,501 cases of pneumonia there were but ten cases of meningitis." These numbers are equivalent to rates of 0·67 per cent. of the total cases, and 2·8 per cent. of the cases in which a post-mortem examination was made.

Pericarditis has been recorded in 9·74 per cent. of the pneumonia autopses here analysed. Roughly speaking, one-third of them were instances of purulent pericarditis. As noted in the table, a certain number of cases classified as serous pericarditis may not, strictly speaking, be properly included under this heading, as the pericardium itself was not obviously inflamed, although the pericardial sac was distended with fluid. Musser & Norris[6] state, that acute pericarditis was observed 268 times in 2,128 autopsies (12·6 per cent.). In 38 cases complicating pneumonia, recorded by G. A. Scott,[6] the exudate was sero-fibrinous in 52·6 per cent. and purulent in 44·7 per cent. In six out of twenty-two cases recorded by Musser & Norris the pericarditis was fibrino-purulent, a percentage of 27·2. The figures of both Scott and the latter authors are small; and the value of the percentage of purulent cases in our returns lies between the values obtained from their figures; we cannot say, therefore, that this form of pericarditis is either more or less common among these Natives than among Europeans. As regards the total number of cases of pneumonia complicated by pericarditis irrespective of the type it would seem probable that pericarditis is rather less common among "Tropical Natives" than among Europeans.

Pleurisy. It is perhaps doubtful whether this condition should be considered as a complication of pneumonia. Musser & Norris write:— "Pleuritis occurs in practically all the cases, or at least in those cases in which the inflammatory process reaches the surface of the lung." Amongst autopsy records from 974 cases they record pleural effusions as occurring in 41·58 per cent., which corresponds closely with the percentage found in Table XVI. It is worthy of note that the pleural effusion with adhesions are sometimes found on the opposite side of the chest to that containing the consolidated lung.

Consolidation of the Lung. In regard to the lobe affected the following distributions may be compared with that recorded in Table XVI.

TABLE XVII.

Lobe affected.	Aufrecht per cent.	Norris. per cent.	Musser & Norris. per cent.	Table XVI. per cent. ‡
Right Upper	9·6	11·0	11·1	7·75
,, Middle	2·3	2·0* (3·0)	2·2	0·91
,, Lower	22·6	29·0	27·8	9·88
,, Two lobes	7·5		6·3	10·49
,, Three lobes	10·0	9·0	8·2	14·89
,, Total	52·0	51·0* (52·0)	55·6	43·92
Left Upper	3·9	4·0	4·5	2·13
,, Lower	24·8	23·0	24·6	11·70
,, Both	6·7	7·0	7·6	10·33
,, Total	35·4	34·0	36·7	24·16
Both Lungs	12·6	21·0* (13·0)	7·5	31·91
Number of cases	1501	493	4062	658

* Percentages areas given in the original, figures in brackets are rates calculated from the actual figures recorded.

‡ Rates given here are not identical with those of Table XVI., as the 30 cases in which the lung affected was not specified have been omitted.

Adami,[8] quoting Orth, states that the right lung is affected in fifty-two per cent. of cases, the left in thirty-three per cent., and both lungs in fifteen per cent. of cases. In the figures from the Native cases we find both lungs are much more commonly affected than in any of the examples quoted, and also that the right lung is rather more frequently attacked than is the case among European patients. Further, among the Natives the disease is not so commonly limited to one lobe: this may indicate that the Native is less resistant to the extension of the infective process than the European.

The appearance of the lung as seen at the necropsy in regard to the " stage of hepatization " does not bear a constant relationship with the length of illness before death. In Table XVIII. the stage of the portion of the lung in the most advanced condition is recorded.

This table illustrates the fact that the " stages of hepatization " of the lung are very variable in the time of their appearance. The lung may be found in the stage of " grey hepatization " within a day or two of the apparent onset of the symptoms, or may still be in the " red stage " after ten or more days of illness. In the earlier part of the table there are no doubt a certain number of cases where the date of admission does not correspond even approximately to the com-

TABLE XVIII.

Appearance of the Lung at the Necropsy.

Day of death after admission.	Acute congestion. Commencing consolidation.	HEPATIZATION.				Resolving.	Normal.	Total.
		Early Red.	Red.	Red to Grey.	Grey.			
1— 5	12	21	38	47	65	5		188
6—10	1	10	44	57	77	7		196
11—15			9	18	39	12		78
16—20		3	3	2	16	4		28
21—25			2	4	10	1	1	18
26—30					7	5	4	16
31—40		1	1	1	6	2		11
41—50				2	7	1	1	11
51—60					1		1	2
61—70						1	1	2
71—80						1		1
	13	35	97	131	228	39	8	551
Percentages	2·4	6·4	17·6	23·8	41·4	7·1	1·4	

TABLE XIX.

Day after admission to hospital on which death occurred.

Day after admission.	No. of deaths.	Day after admission.	No. of deaths.	Day after admission.	No. of deaths.	Day after admission.	No. of deaths.
1	29	8	56	15	16	46— 50	5
2	32	9	39	16—20	38	51— 55	1
3	52	10	27	21—25	24	56— 60	1
4	63	11	27	26—30	21	61— 65	1
5	60	12	27	31—35	14	66— 70	2
6	54	13	23	36—40	6	71— 80	1
7	62	14	16	41—45	10	81— 90	0
						91—100	2
						Total	709

mencement of the illness, and in the latter part it may be an attack in a second lobe which has been noted in the post-mortem report, when the first lobe to be affected may have become normal in appearance. Even when reasonable allowance has been made for these sources of error we still see that the rate of change in the lung tissue, as judged by the general appearance, must vary within wide limits; and thus, in any individual case, the stage of hepatization is but little guide to the length of illness before death.

Chart X. represents the number of deaths occurring on each day after admission to hospital. The numbers are given in Table XIX.
~~table~~ :—

This distribution is almost certainly heterogeneous, and cannot, I find, be fitted by any single "Pearson curve." If we eliminate a portion of the tail as shown on the chart by the dotted line, an approximation to the bulk of the distribution may be obtained; giving the mean period before death as 7·5 days, and the modal period as three days. Further adjustments have been attempted without any very marked improvement in fit, and it would appear almost impossible unless unreasonable alterations are made, to increase the position of the mode to more than about 3·8 days. An explanation of the want of fit of the original distribution may be that the later deaths are not due, strictly speaking, to pneumonia, but rather to some super-added infection, for example by a streptococcus, and if this had not occurred the pneumonia would not have been fatal. If this be the explanation we could obtain an approximate measure of the number of deaths due to intercurrent or subsequent infections. Further study may lead to a more satisfactory analysis of this distribution, but there is little doubt that it is heterogeneous in character and does not represent deaths due solely to pneumococcal infections.

SECTION III.

A Consideration as to whether there be any Evidence that Pneumonia is an Infectious Disease.

Before any discussion of the question whether pneumonia may be spread from case to case can be usefully undertaken it is necessary to consider the meaning of the terms "Infectious" and "Infective." Considerable confusion seems to exist at present in medical circles as to what these terms respectively connote.

If we turn to the definition of *Infectious* in the New Oxford English Dictionary we find the following:—

"1. Having the quality or power of communicating disease by infection; infecting with disease; pestilential; unhealthy."

"2. Of diseases: Apt to be communicated or received by infection; liable to be transmitted from one person to another by means of air or water."

Under *Infective* we find:—

"1. Having the quality of affecting injuriously or tainting." . . .

"This is the old and almost obsolete use."

"2. Having the quality of infecting with disease, or spreading disease by infection; infectious." . . . . .

"Modern use."

It would appear from these definitions that the modern use of Infective is as a synonym for Infectious.

In Fagge & Pye Smith[10] we find the following statements:— "From an early period it has been observed that certain diseases are contagious or 'catching,' either by direct contact with the sick person, or by means of clothing or buildings, or by conveyance through the air of a presumed *materies morbi:* When actual contact was necessary to reproduce it the disease was called *contagious;* when it was transmissible by the air it was given the wider name of *infectious*. . . . The distinction is not fundamental."

"The word *infective* has been used of late to denote processes which spread, not always from one person to another, but from one tissue or organ of the body to another."

Here we have a definition of infective which is not synonymous with infectious, as some infective diseases are not infectious.

Notter & Firth[11] write as follows:—

"This conception of the origin of the causes of disease if interpreted literally, implies the belief that every single case of each infective disease is the offspring or result of an antecedent case of the same disease. A little reflection and experience soon indicate that the doctrine is too inelastic for general acceptation, because certain of these diseases affect the lower animals as well as man, and consequently the antecedent case of a given instance of such disease need not be a human case; while as regards the others, which are known to affect man only, the pre-existing case must be sought in man alone." From the same authors, under the heading "The Infective Diseases," we find the following:—"According to the manner in which these diseases are transmissible from one person to another, so are they spoken of as being either infectious, contagious, or inoculable."

Here again we have infectious diseases regarded as a sub-group of infective diseases, but in the first quotation from the same authors we are told that the source is not always a human case of the disease, and we must therefore include diseases derived from animals. Are we then to exclude from the term infective diseases cases of bacterial infection which arise, not from any previous case, but from an organism which was previously a saprophyte,* for example some members of the coli group? Or again, as in the case of tetanus, where the invading organism is not derived from a previous case of the disease, or at least where such connection cannot be traced.

We find that Sir W. Osler[7] classifies under the heading "Specific Infectious Diseases" the following sub-groups:—

A. Bacterial Diseases.

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* "Parasite" would be possibly a better term to use in this connection than "Saprophyte." It almost seems as if a new term were wanted to describe an organism which can exist and multiply on the surfaces of its host without giving rise to any abnormal symptoms, but which can become pathogenic under altered conditions. Whether this change from "harmless parasite" to "pathogenic parasite" be due to alterations in the host, or to alterations in the type of the organism, or to both factors together, is a matter of conjecture. I believe, the most likely hypothesis is, that an alteration in type occurs which is usually occasioned by some antecedent alteration in the fluids or tissues of the host. That members of the coli group may take on pathogenic characters is established, and it is possibly true also of members of other groups; for instance, the diptheroid group, and the tubercle or "acid fast" group. It also seems to me probable that a "pneumonia" group exists, and that we shall come to recognise the different strains of pneumococci as being as separate in character as the members of the coli group, and perhaps as even more easily altered in character. Dr. F. S. Lister's recent work in this Institute, which will shortly be published, appears to support this view, and if confirmed will place on a much more solid basis what is at present merely a speculation. The presence of the pneumococcus in the throats of the apparently healthy, its association with epidemic outbreaks of acute naso-pharygeal catarrh, its causal relationship with lobar pneumonia, the marked differences between various strains in their effects on experimental animals, and the rapid alterations which often occur under laboratory conditions—all these and other facts point to the possibility or even probability that the organism known as Fraenkel's pneumococcus is not a single entity but rather a group of pneumococcal organisms. This question is referred to here because it has a bearing on the problem of infection.

B. Fungus Infections, not bacterial.
C. Protozoan Diseases.
D. Diseases due to Metazoan Parasites.
E. Infective diseases of doubtful or unknown etiology

Under " A " we find tetanus, and it is therefore classed as an infectious disease, and in the sub-group infective diseases we find smallpox. The term " infectious disease " then apparently corresponds to Notter & Firth's " infective disease," and is in disagreement with the definition given by Fagge & Pye Smith. But on page 78,7 we find this sentence:—" Clinically, the *infectious nature* of pneumonia was recognised long before we knew anything of the pneumococcus." And further:—" As many as ten occupants of one house have been attacked." This latter remark, in its relation to pneumonia, will be discussed later on. Thus " clinically infectious " has clearly a different meaning to " infectious." If we start by defining all bacterial diseases as infectious, then having once shown that a disease is causally connected with a bacterium it is unnecessary to discuss whether or no it be infectious.

This classification has, to my mind, several objections; to make the word " *infectious* " practically synonymous with " *due to a micro-organism* " in respect to diseases, does not help us to a more accurate method of thought, and has, moreover, the serious disadvantage that it changes the ordinary, or at least the older, meaning of the word. This change has led, and will lead to constant misunderstanding and confusion, and the necessity of introducing the expression " clinically infectious " exemplifies the author's need for a new term to correspond with the old usage of the word infectious with its meaning of " catching."

Let us turn now to definitions given in text-books of Infectious Diseases, for the term when thus employed has an old and well established meaning, totally different, however, from that used by Sir W. Osler. Ker,12 in his admirable text-book, defines infection thus:—" By infection is understood the invasion of the tissues by a pathogenic bacterium. . . . And here it may be remarked that it is difficult to draw a hard and fast line between pathogenic and saprophytic bacteria." . . . " It is, with our present knowledge of the bacterial causes of infectious diseases, difficult to draw fine distinctions between contagion and infection. . . . A pathogenic organism, however, may be regarded as one capable of producing disease in man under natural conditions." Here we are left wondering what the phrase " natural conditions " may imply, and whether we are to exclude from the classification " pathogenic bacteria " those which produce disease in animals but not in man.

The following definition is given by Goodall & Washbourn[13]:—"Diseases which are transmissible from one individual to another are said to be *contagious* or infectious. Strictly speaking, the term infectious is applied to diseases transmissible through the air, while the term contagious is used of diseases which are spread by direct contact, or by infected articles." In this definition we have returned to a definition more like that given by Fagge & Pye Smith, and more in accordance with the general or "lay" use of the word.

It is surely meaningless to speak of one disease as more or less infectious than another, or to use such an expression as "highly infectious" for a disease like small pox, if we are going to use the word as synonymous with bacterial, in Sir W. Osler's terminology; but there is clearly a need for some term corresponding to the term infectious as employed and defined by Goodall & Washbourn.

Historically, it is not difficult to see how the extension in the use of the term may have taken place; but whether such an extension is scientific, that is whether it helps us to classify the facts more accurately, is perhaps open to doubt. Some argument such as the following may have been used—"catching diseases" were in some instances proved to be due to micro-organisms, therefore, by analogy all such diseases are probably dependent on some micro-organism. The micro-organisms of disease are specific, and therefore the organisms in one case must have been obtained directly from those of a previous case. This must be true therefore of all diseases due to micro-organisms; and thus all such diseases are infectious, and we should, logically, extend the term "infectious diseases" to the whole group of complaints due to micro-organisms, and even further, to those of metazoal origin.

If this were the argument employed it is clearly unsound. A series of assumptions are made which have not been proved; they are, however, assumed to be true, and a generalisation drawn which has had the effect of leading the student to regard the question as settled This tends to obscure the need for a patient search for the evidence which shall prove or disprove them. However useful these hypotheses may be in their legitimate sphere, they are dangerous when interpreted as reliable theories; and moreover, grave doubt has already been thrown on the validity of some of them. Saprophytic organisms have been shown to acquire pathogenic properties, and thus an antecedent case of the disease need not necessarily be postulated.* The specific character of several pathogenic bacteria is also seriously in doubt. That a very large number of pathogenic bacteria may live as saprophytes is proved by the varieties which can be grown on artificial media, and although it may be reasonable to assume that similar conditions are not paralleled in Nature this has not been established as a fact.

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* If for the sake of argument we assume that healthy people communicate the pneumococcus to others who also remain healthy, so that a large number of the population harbour the organism as a harmless parasite, it would serve no useful purpose from a practical point of view to class pneumonia as an infectious disease; unless indeed the organisms spread by the sick had different properties, i.e., were virulent to the healthy, in which case evidence of this should be found in the statistics.

All these questions require careful investigation with an open mind, and any phraseology which implies that they have been proved is, it seems to me, retrograde. If this be admitted, the need for a word which will express the common meaning " catching " and will also connote a definite train of events formerly covered by the word infectious, is evident.

Considering the very old established use of this word it would not seem wise to abandon it and further complicate the terminology by the introduction of another, despite the fact that one of the leading text-books of Medicine has extended the meaning as described above.

It may be considered by some that the term " communicable " would be suitable, seeing that there is a growing tendency to use this word in place of infectious. It seems to me that this word, used in the sense "infectious," is not free from ambiguity. It suggests that the disease is capable of being communicated, rather than of communicating itself; that is to say, a suggestion of a deliberative agent is introduced. Thus, Vaccinia in man is a communicable disease, but scarcely infectious in the sense in which it will be defined here. Again, the term " catching " might be used as an alternative; but it is also liable to misuse, and does not seem suited to accurate thought; for we constantly hear such expressions as " catch a chill " or " catch a cold " where the suggestion of contact with a previous sufferer is not intended. On the whole, then, it would appear to me better to retain the term " infectious " and try to define it so as to make its use free from ambiguity.

The following definition of an infectious disease is therefore suggested:—*An " infectious disease " is one which may be spread from the affected to the healthy, and is such that the healthy person incurs a risk of contracting the disease by associating with a person suffering from it, either directly, or indirectly through the medium of clothing, furniture, food-stuffs, etc., which have been contaminated by the sick.* It may be objected that a disease spread by healthy persons, that is by those who have never suffered or exhibited any symptoms of illness, should nevertheless be classed as infectious. Such a disease would only be excluded from the definition infectious if those actually showing symptoms did not spread it, or did not constitute any increased risk to the population; that is to say, that those in their immediate neighbourhood did not, on the average, suffer more than people at a distance. From the practical point of view, it seems improbable that any administrative steps could be taken to deal with a disease which spreads as commonly from the healthy as from the sick; but, if acquired more commonly from the sick, it will fall within the limits of our present definition, and so statistical evidence should be forthcoming in proof of such a fact.

It will be seen, therefore, that the proposed definition is purely utilitarian, and if accepted we have a right to ask for proof that association of the healthy with the sick constitutes a risk to the former, before we consent to classify any disease as infectious.

In regard to diseases spread through animals, three classes must be considered:—

Firstly. Diseases in which the animal is itself affected, and in the spread of which man *need* play no part; e.g., Tuberculosis or Glanders.

Secondly. Diseases in which the animal (usually an insect) is necessarily the intermediate host, e.g., Malaria or Tick Fever.

Thirdly. Where the animal concerned (again usually an insect) is merely a mechanical carrier of infection, e.g., Bubonic Plague.

The first group will fall within the terms of the definition, if, when spread to man he in turn infects his neighbours. Possibly a special category must be made for a few parasitic diseases where man and an animal are alternate hosts. The third group will necessarily be classified as infectious because (insect) carriers will be more numerous in the immediate neighbourhood of the diseased. In regard to the second group, and to a less extent the third also, a problem arises of considerable practical importance. It will be evident that when an special animal host is essential to the spread, that the disease will only become infectious when the intermediate host is present. Thus a disease may be infectious in certain regions and cease to be infectious when imported into others; or again, it may be infectious only at certain seasons. This is a distinction of some practical importance.

Further, it is conceivable that a disease might be infectious among one race and not among another; habits and customs might be determining factors in its spread, or constitutional immunity might exist. For instance, a race may be conceived as having developed through selection an immunity, and the introduction of a case, therefore, into such a community would not be a source of danger. In such circumstances this disease could not reasonably be classified as infectious in respect to such a population.

It is evident, then, that this definition is purely a practical one, but from an administrative point of view is, it appears to me, of importance; and further, it is, I think, a reversion to the older and more commonly accepted use of the word. In the following para graphs the word "infectious" will be used with the meaning given to it in the above definition.

Many authors, from v. Jürgensen in 1874 to those of the present day, have claimed that pneumonia is an infectious disease, not in Sir W. Osler's sense that it is due to a micro-organism, but apparently in the sense in which the word has just been defined. Musser and Norris[6] write as follows:—"To sum up, we are forced to the conclusion that pneumonia is a transmissible disease, to be classified with the infectious fevers, and like them to be guarded against by the application of antiseptic principles." They subsequently proceed to describe the pneumococcus of Fraenkel as *the micro-organism of lobar pneumonia*, and thus under this term they are dealing apparently with a specific disease, and not merely with a group of signs and symptoms. Why then, we may ask, do they include the "Middlesborough Epidemic" among the list of examples quoted to indicate that pneumonia may be spread by infection, when the organism described by the investigators of this outbreak was not the pneumococcus? No evidence in favour of the spread by infection from case to case can be considered valid if the identity of the associated organism be not determined. Further, they state that in Kimberley this disease is limited to the Kaffirs; how such a curious misstatement could have arisen it is difficult to understand, but in any case evidence must surely be much more carefully sifted, than appears to have been done by these authors, before valid conclusions can be drawn.

The "Middlesborough Epidemic," so often quoted in support of the contention that pneumonia is an infectious disease—and amongst others by Notter & Firth[11]—shows that outbreaks of a disease similar to, but not identical with, pneumonia may occur; we must, therefore, be particularly cautious before concluding that an epidemic is due to the pneumococcus in the absence of bacteriological proof. In most of the instances quoted by the authors of text-books no mention of the organism associated with the disease in question is made, and there is therefore no direct evidence that the disease was due to the pneumococcus of Fraenkel, which organism we are assuming to be the cause of Pneumonia. This is a criticism which must apply to all evidence collected prior to the recognition of this diplococcus; and it also applies, so far as I know, to the instance of ten cases in the same house, already quoted from Sir W. Osler's book. This, however, is not the only exception which must be taken to this example when adduced as a proof of infection.

To prove the infectious nature of any disease it is not necessary merely to collect examples of runs of cases in one house or family; we must show that such runs occur more frequently than they should do on the laws of chance, supposing every similar individual in the community equally liable to contract the disease, whether he be, or be not in contact with one already affected. So far as I know, no attempt up to the present has been made to follow such a method in regard to pneumonia.

Our figures deal entirely with the African Native from "tropical areas," and any conclusions which we draw from them in regard to the problem of infection must not be applied unreservedly to Europeans in South Africa, much less to Europeans in Europe. A careful statistical analysis of European data, is, I believe, the only way in which these problems with regard to the latter groups can be satisfactorily settled, and the argument from analogy, if used at all, must be employed with extreme caution.

In the "8000 experiment," records of the medical history of all the Natives included were kept in each case for six months, and in some for longer periods. All these Natives passed through the Witwatersrand Native Labour Association's Compound, where they were given serial numbers. On arrival in the compound the "boys" line themselves up under Captains, and those coming from the same district stand together. As a consequence relations, friends, and acquaintances will, on the whole, tend to carry consecutive numbers. On allotment a group under any Captain is not broken, so that subsequently on the mine "boys" carrying consecutive numbers are in closer association than those with more distant numbers. In Portuguese territory a similar method of numbering is adopted, and it appears that the order in which the "boys" obtain their numbers, although not identical with the W.N.L.A. order, is much the same; in practice one finds little difference in the results calculated from either the Portuguese or W.N.L.A. Depôt Numbers.

If we suppose that one case of pneumonia in a community of Natives does not increase the risk to the others, we may expect that the various cases of this disease will not be unduly grouped. Grouping might, nevertheless, occur owing to a variation in the prevalence of the disease on different mines; a point which will be discussed later. It will be noted that runs of twos, threes, and so on, are to be expected even on the assumption that the "laws of chance" alone determine the distribution. By runs or sequences, we mean affected Natives carrying consecutive numbers. If, however, the disease tends to spread from the sick to the healthy, that is, it is infectious, then we should expect that runs of twos, threes, etc., will be more common than if chance were the only factor concerned.

By means of the following formula we can calculate the most probable chance distribution for the appearance of sequences.

Let N = number of "boys" in the series,

m = number not attacked,
n = number attacked,

then it can be shown that the various terms of the following expression will represent the most probable distribution for the number of sequences,*

$$\frac{m\,(m-1)}{N-1}\left[1+\frac{n}{(N-2)}+\frac{n\,(n-1)}{(N-2)\,(N-3)}+\frac{n(n-1)\,(n-2)}{(N-2)(N-3)(N-4)}+\cdots\right]$$

The successive terms can be easily calculated from the preceding one; thus, if R_0, R_1, R_2 etc., represent the terms, and if

$$R_0 = \frac{m\,(m-1)}{N-1}$$

$$R_1 = \frac{n}{N-2}R_0$$

$$R_2 = \frac{n-1}{N-3}R_1 \quad \text{etc.}$$

Now, even if the laws of chance alone are operative in determining the distribution, we must not expect that the experimental numbers will be identical with those of the most probable distribution; but we can, by Pearson's method of "Goodness of Fit,"14† find the chance that the observed distribution has arisen owing to errors of random sampling.‡

Let us now turn to the actual results from 8,088 Natives ("8000 experiment"). Regarded as one group, we find:—

N = 8,088, m = 6,988, n = 1,100.

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* The first term represents a zero sequence, i.e., where two unattacked "boys" come together, the second a sequence of one, i.e., where there is an attacked "boy" with an unattacked "boy" on each side, the third a sequence of two attacked between unattacked, and so on.

† I am indebted to Dr. Troup for most of the mathematical analysis in the proof of this problem, and although it is not yet free from difficulty, the matter is too technical for discussion here. We hope to publish it elsewhere shortly.

‡ The meaning of this will be made clear if we consider a simple example. If we toss ten pennies, the most probable result will be five heads and five tails; that is to say, if we repeat the experiment 1,000 times we shall find five heads and five tails to be more common than any other result. But we shall find that four heads and six tails, three heads and seven tails, and so on, also occur. If, therefore, on the first throw we find three heads and seven tails, we must not straightway assume that the coins are biased. If we knew nothing about our coins we should then proceed to calculate the probability of such an event occurring on the assumption that they were not biased, and our result would be in the form P = x where x is the probability that such a divergence would occur on the laws of chance. Thus if the probability is P = 0·2 we can say such a divergence from the most likely will, on the average, arise twice in every ten such examples.

TABLE XX.

Sequences of	Theoretical number.	Observed number.	
1	821	768	
2	112	122	
3	15	16	
4	2	8	
5	0·31	1	
6			
			P=0·00004

Here we have a defect in sequences of one and an excess over theory at the higher groups; and considering the value of "P" we may conclude that it is highly unlikely that a distribution such as this has arisen owing to errors of random sampling. It is possible, however, that the heterogeneous nature of this population may be responsible in part for the value found. This group has, therefore, been sub-divided according to the territorial areas from which the Natives were recruited.

TABLE XXI.

Mozambique Natives.

Groups.	Theory.	Observation.		
1	231·86	214	N=2304	
2	31·03	34	m=1995	
3	4·15	5	n= 309	
4	0·66	3		P=0·017

Here only seventeen times out of 1,000 could such a divergence from theory be expected on the laws of chance.

TABLE XXII.

Angoche or Parapat Natives.

Groups.	Theory.	Observation.			
1	98·11	85		N= 889	
2	15·06	15		m= 752	
3	2·30	3		n= 137	
4	·38	1	2		
5		0			
6		1			
					P=0·020

Here only twice in a hundred trials is a fit as bad as this to be expected.

TABLE XXIII.

Quilimanes.

Groups.	Theory.	Observation.		
1	121·05	128	N=1141	
2	17·45	13	m= 976	
3	2·50	1	n= 165	
4	·39	2		P=0·028

TABLE XXIV.

Portuguese Nyassas.

Groups.	Theory.	Observation.		
1	232·81	210	N=2171	
2	34·26	42	m=1851	
3	5·03	6	n= 320	
4	0·88	2		P=0·148

In this case, as in that of the Mozambiques, the ones are in defect, the twos, threes, and fours in excess.

TABLE XXV.

Tete & P.I.T.

Groups.	Theory.	Observation.		
1	140·13	139	N=1572	
2	15·81	18	m=1394	
3	1·77	1	n= 178	
4	0·20	0		P=0·842

Here we could not expect a better agreement between observation and theory on the laws of chance.

Taken together, these tables show in a high degree, the improbability that the observed deviations from theory have arisen by chance; but we must not necessarily assume that the deviations are therefore due to infection.

Considering the conditions under which the Natives live and work on the mines, the chance that an affected "boy" will spread the disease, if it be infectious, to a "boy" carrying a neighbouring number, will not be much less than the chance of his spreading it to a consecutive number, while the likelihood of his conveying it to a "boy" with a distant number is much more remote.

If then the disease be spread from case to case we might expect to find—if we reverse our problem and calculate the most probable distribution of the runs of unaffected "boys"—that the small runs, of say ones, twos, three, and also the very long runs, will be in excess of theory, with the consequence that the runs of intermediate size would be in defect.

To obtain the theoretical distributions for the new series we must re-write our formula, thus:—

$$\frac{n(n-1)}{N-1}\left(1+\frac{m}{(N-2)}+\frac{m(m-1)}{(N-2)(N-3)}+\cdots\cdots\right)$$

Here the first term or the sequences of 0 is of importance. A sequence of 0 now means two affected "boys" with no unaffected "boys" between. Thus a run of three affected "boys" is equivalent to two sequences of 0, and so on. The value of the probability for the whole group of 8,088 Natives is $P=0\cdot002$ approx. The 0's are considerably in excess and supply 8·19 to the value of χ^2*[14], the 1's supply 0·94, and the 3's 1·04, the 2's are slightly in defect, 7's, 8's and 10's are considerably in defect, contributing together 12·63 to the value of χ^2. Excesses are again found at the tail of the distribution.

For the Mozambiques (23 groups), $P=0\cdot33$ approx. The χ^2 value was not calculated for four runs at the tail, namely, one each of 43, 46, 53 and 71; these will decrease the value of P, but the labour of carrying the calculation to 71 terms would be hardly justified by the increased accuracy to be obtained. Here runs of 0's and 1's are in excess, but otherwise no very marked abnormality in the distribution occurs. After 25 the tail shows errors in excess.

The same general statement is true of the Angoche Natives, but marked divergence from theory occurs in the 0's, which group contributes 4·77, and also in the groups 18 to 20, which together contribute 7·81 to the χ^2 value, $P=0\cdot07$.

Table XXVI., page 53, is given as an example of the method. Differences are calculated only to nearest whole number in the column headed "Theory."

A similar table worked from the Portuguese numbers gave $P=0\cdot65$.

The distribution for the Nyassa's leads to, $P<0\cdot02$. In this distribution there is a marked excess in the groups 0, 1, 2 and 5, and a marked defect in groups 7, 8, 9, 10 and 11; excesses again appear after 24.

Among the Tete & P.I.T. Natives excesses are found in the groups 8 to 14, and defects in 2, 4, 5, 6 and 7, and the tail of the distribution

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*The meaning of this symbol will be found in the essay of Professor Pearson, already referred to.14 It is the sum of the differences of the theoretical and observed values squared and divided by the theoretical value. Thus if m = observed value, and m^1 = theoretical value, then $\chi^2 = S\left[\frac{(m-m^1)^2}{m^1}\right]$.

TABLE XXVI.

Quilimane Group.

Group.	Theory.	Observation.	Obs.—Theory.	χ^2
0	23·74	21	—3	0·3791
1	20·34	24	+4	0·7866
2	17·43	11	—6	2·0654
3	14·93	15	0	
4	12·79	19	+6	2·8507
5	10·95	9	—2	0·3653
6	9·38	9	0	
7	8·03	10	+2	0·4981
8	6·87	5	+2	0·5822
9	5·88	6	0	
10	5·03	5	0	
11	4·30	6	+2	0·9302
12	3·68	5	+1	0·2717
13	3·15	3	0	
14	2·69	4	+1	0·3717
15	2·30	4	+2	1·7391
16 to 20	7·34	3	—4	2·1798
21 to 25	3·34	2	—1	0·2994
26 to 30	1·54	3	+1	0·6494
31 to 45	1·28	1	0	
				13·9687

P=0·78

is not markedly, if at all, in excess of theory. P=0·11 approx.

These results are summarised in Table XXVII.

TABLE XXVII.

Summary of results in Tables XXI. to XXVII.

Total.	Value of "P" for sequences of Pneumonia.	Value of "P" for sequences of unattacked Natives.
Total	0·00004	0·002
Mozambique ...	0·017	0·33
Angoche ...	0·032	0·07
Quilimanes ...	0·028	0·78
Nyassas	0·148	<0·02
Tete & P.I.T. ...	0·842	0·11

We thus reach the conclusion that if every "boy" within a group has an equal chance of contracting pneumonia—which of course is one of the assumptions which must be made if the distribution is to be described by the laws of chance—then, with two or possibly three exceptions, we can assert that the observed divergencies are unlikely to have arisen by chance.

But infection from case to case is not the only cause which might bring about this result. It is quite conceivable that certain sub-groups within the territorial groups may be more susceptible than others. We know that in regard to race, altitude, and climate of their homes, no group is even approximately homogeneous. Before making any assumption as to the cause of the divergencies we must study the matter more in detail.

It may here be interesting to record that the values for "P" in the case of a Chicken Pox distribution amongst the Quilimane Natives (for the same group of Natives as in Table XXVI.) was found to be zero to more than six places of decimals; and that the χ^2 values for both tables, viz., that grouped according to sequences of cases of Chicken Pox and sequences of the unattacked, is larger in proportion to the number of sub-groups than any of the values for the pneumonia distributions. In the Chicken Pox distribution (the unattacked), two runs occur, one of 150 and the other of 445; these are very considerably in excess of any found in the pneumonia distributions.

The dates of admission to hospital of the Natives suffering from pneumonia who constitute the runs of threes, fours, and six, are given in Table XXVIII. and XXIX. The small numbers between the dates show the number of intervening days.

We notice on reference to the table of "sequences of threes" (Table XXVIII.) that on only two occasions were all the cases admitted to hospital within thirty days. Among the groups of four and over there are three runs of threes admitted within this period, and none of four and over. Further, we note that a considerable number of pairs occurred in these groups with very short intervals between the admissions; this, if we assume infection from case to case, would imply a very short incubation period—unless both cases were infected from some common source and not the one from the other—whereas in the runs of threes we must assume a much longer period. The incubation period of most infectious diseases varies within fairly narrow limits, in the absence, therefore, of definite information each observer will, for the present, have to decide for himself what range he considers it reasonable to assume as a working hypothesis in respect to lobar pneumonia.

To return to a subject previously mentioned, namely, the varying prevalence of pneumonia on different mines. It is quite possible that there may be some factor or factors operative to a different extent on

TABLE XXVIII.

Table giving dates of admission to hospital of Natives bearing consecutive numbers in sequences of three.

Mozambique.	Angoche.	Nyassa.	Quilimane.
28.10.12	18. 9.12	28. 8.12	14. 9.12
114	*71*	*14*	*74*
19. 2.13	28.11.12	11. 9.12	27.11.12
42	*5*	*48*	*96*
2. 4.13	3.12.12	29.10.12	3. 3.13
8. 9.12	30. 9.12	6.11.12	
59	*52*	*5*	
6.11.12	21.11.12	11.11.12	
40	*162*	*39*	
16.12.12	2. 5.13	20.12.12	
			Tete.
23.11.12	30.10.12	16. 8.12	13.11.12
31	*2*	*1*	*23*
24.12.12	1.11.12	17. 8.12	6.12.12
27	*138*	*150*	*39*
20. 1.13	19. 3.13	14. 1.13	14. 1.13
23.11.12		1. 2.13	
0		*10*	
23.11.12		11. 2.13	
136		*16*	
8. 4.13		27. 2.13	
22.11.12		16.10.12	
61		*55*	
22. 1.13		10.12.12	
14		*79*	
5. 2.13		27. 2.13	
		16.11.12	
		21	
		7.12.12	
		3	
		10.12.12	

TABLE XXIX.

Table giving dates of admission to hospital of Natives bearing consecutive numbers in sequences of four and over.

Mozambique.	Angoche.	Nyassa.	Quilimane.
22.11.12	16. 2.13	26. 8.12	23.10.12
0	*22*	*0*	*5*
22.11.12	10. 3.13	26. 8.12	28.10.12
1	*25*	*78*	*115*
23.11.12	4. 4.13	22.11.12	20. 2.13
69	*3*	*3*	*28*
31. 1.13	7. 4.13	25.11.12	20. 3.13
4.10.12	22.11.12	31.10.12	11.10.12
39	*35*	*27*	*30*
12.11.12	27.12.12	27.11.12	10.11.12
6	*65*	*1*	*42*
18.11.12	1. 3.13	28.11.12	22.12.12
17	*12*	*135*	*47*
5.12.12	13. 3.13	12. 4.13	7. 2.13
	14		
	27. 3.13		
	32		
	28. 4.13		
16. 9.12			
126			
20. 1.13			
70			
31. 3.13			
24			
24. 4.13			

one property as compared with another; this would cause an undue grouping of cases when they are examined by the methods just adopted. An estimate of the effect of this factor may be arrived at in the following manner. We may regard each series of twenty-five "boys" as a group by itself, and determine how many cases of pneumonia occurred in each group. We can then calculate how many of these groups should have had no cases of pneumonia, how many one case, and so on, by the formula[20]:—

$$p_s = \frac{m\,(m-1)^{n-s}\,n\,!}{s\,!\,(n-s)\,!\,m^n}$$

TABLE XXX.

Number of cases of pneumonia occurring in groups of 25 Natives with consecutive numbers.

Number of Cases.	Observed frequency of occurrence.	Theoretical frequency.	Difference.
0	16	10	+ 6
1	44	36	+ 8
2	55	61	— 6
3	58	70	—12
4	69	60	+ 9
5	30	41	—11
6	23	24	— 1
7	14	11	+ 3
8	7	5	+ 2
9	1	2	— 1
10	2	1	+ 1
11	2	0	+ 2
	321	321	

It will be noticed that while considerable differences are found between the observed and the expected frequencies, the plus and minus differences alternate, no definite order being maintained.

If infection from case to case were the cause of the departure of the observed from the theoretical frequency, we should expect the groups with the larger numbers of affected to be considerably in excess, while those with the smaller numbers should be uniformly in defect; this, however, is not so. This method of dealing with the problem is not entirely free from objection, nevertheless it should, it seems to me, be sufficiently accurate to demonstrate any general law underlying the grouping should such exist.

Two other sources of error also exist; firstly, all the Natives comprised in the foregoing tables were not under observation for the same length of time; and secondly, the attack-rate is a function of the period of residence on the Reef. Allowances for both these sources of fallacy will be made in the following analysis.

We may now consider this problem from another point of view. If the pairs arise entirely at random, then the interval between the dates of attack—or what we have to consider in our data, the dates of admission to hospital—will also form a chance distribution; that is to say, the number of pairs observed with any particular interval between the dates of admission to hospital, will, within the limits of errors from random sampling, be proportional to the chances of pairs with such intervals occurring. Now it is clear that, if the period is limited to 180 days, a pair of 0's (i.e., both members of the pair admitted to hospital on the same day) may occur on 180 days, but a pair with a 179 days interval can only occur once, as the first member of the pair must be attacked on the first day of observation and the second on the last, so that the chance of a pair with an interval of 179 days would be 180 times as unlikely as a pair with no days interval.*

This statement would be strictly true only if the chance of contracting the disease was uniform throughout the period, and if no gaps were made in our series by deaths and repatriations. The former difficulty will be discussed more at length later, and the latter must be borne in mind when we come to interpret the deductions which can be drawn from these figures. This factor will also tend to reduce the total number of pairs recorded.

Table XXXI. gives the number of pairs tabulated according to days of interval between admission to hospital of each member of the pair, grouped in ten day periods. All the Natives from which these figures were obtained had been under observation for six months, and no pair occurring after this period is included, so that an interval of more than 182 days cannot arise. Groups of three or more Natives bearing consecutive numbers have been split into pairs; thus a group of three is considered as two pairs, a group of four as three pairs, and so on.

A study of this table shows that the "want of fit" of observation to theory is largely due to an excess of cases in the first group. We must, however, remember that the attack-rate is not uniform throughout the period of six months, and we shall obtain a much closer approximation to the true theoretical distribution if we make allowances of the variations in the attack-rate. The following method was suggested to me by Dr. Troup. If we divide the 124 pairs in the same ratio as the monthly attack-rate (see Table III.), we obtain the following groups:—31·9, 25·3, 19·8, 18·2, 16·5, and 12·3. Now let us calculate the distribution of 12·3 × 6 pairs over the whole period,

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* We can calculate the number of pairs to be expected in the following way:—Let n = number of days in the period, then $n + (n-1) + \ldots + 2 + 1 = m = \frac{n}{2}(n+1)$. a = number of pairs. Let it be required to find the number expected with intervals 0-9 days, 10-19 days, etc. Then,

$$T_{0-9} = \frac{a \times 10 (2n-9)\frac{1}{2}}{m} = \frac{10a(2n-9)}{n(n+1)}$$

and $$T_{10-19} = T_{0-9} \frac{2n-19}{2n-9} \text{ etc.}$$

TABLE XXXI.

Table giving days of interval between admission to hospital of each member of a pair as observed, also giving the most probable number and the value of χ^2 for the difference.

Days interval.	Observed number.	Probable number on law of chance.	χ^2
0- 9	25	13·3	10·2925
10-19	12	12·5	0·0200
20-29	16	11·8	1·4949
30-39	17	11·0	3·2727
40-49	10	10·2	0·0039
50-59	8	9·5	0·2368
60-69	5	8·7	1·5736
70-79	6	8·0	0·5000
80-89	4	7·2	1·4222
90-99	6	6·5	0·0385
100-109	2	5·7	2·4018
110-119	5	4·9	0·0020
120-129	2	4·2	1·1524
130-139	3	3·4	0·0471
140-149	2	2·8	0·2286
150-159	1	1·9	0·4263
160-169	0	1·1	1·1000
170-179	0	0·4	0·4000
Total	124		24·6133

P = 0·1043

and (16·5—12·3) or 4·2 × 5 pairs over the first five months, 1·7 × 4 pairs over the first four months, and so on. We shall then, by summing the number of pairs occurring with 0 to 9 days interval, etc., obtain a new distribution which will be the expected distribution when the varying monthly attack-rates have been allowed for. This has been done, and in Table XXXII. the contributions to the total number of expected pairs from each factor are shown in columns 1 to 6. The value of $\chi^2 = 11 \cdot 1725$, which leads to P = ·8, and shows that when the varying monthly attack-rates are allowed for, the distribution observed does not differ from theory by more than might reasonably be expected on the laws of random sampling.

TABLE XXXII.

Table giving the result of calculated distribution of pairs according to the monthly attack-rate.

Interval of days.	COLUMN.						Number of pairs expected by Theory.	Observation.	χ^2
	1	2	3	4	5	6			
0- 9	7·95	2·70	1·08	1·00	3·34	3·62	19·69	25	1·4259
10- 19	7·50	2·52	0·99	0·88	2·74	2·20	16·83	12	1·3714
20- 29	7·05	2·33	0·89	0·77	2·14	0·78	13·96	16	0·2857
30- 39	6·59	2·14	0·80	0·65	1·53		11·71	17	2·4009
40- 49	6·14	1·96	0·71	0·53	0·93		10·27	10	0·0087
50- 59	5·78	1·78	0·61	0·41	0·33		8·91	8	0·0910
60- 69	5·24	1·59	0·52	0·30			7·65	5	0·9467
70- 79	4·78	1·40	0·43	0·18			6·79	6	0·9412
80- 89	4·33	1·22	0·33	0·06			5·94	4	0·3529
90- 99	3·88	1·03	0·24				5·15	6	0·1588
100-109	3·42	0·84	0·14				4·40	2	1·1000
110-119	2·97	0·66	0·05				3·68	5	0·4568
120-129	2·51	0·47					2·98	2	0·3333
130-139	2·03	0·29					2·32	3	0·2130
140-149	1·61	0·10					1·71	2	0·0529
150-159	1·16						1·16	1	0·0333
160-169	0·70						0·70	0	0·7000
170-179	0·25						0·25	0	0·2500
	73·89	21·03	6·79	4·78	11·01	6·60	124·10	124	11·1225

P=0·8497

Unless, therefore, we are to consider that infection from case to case is as likely to occur with an interval of one day as with an interval of several months, there seems to be no justification for the assumption that the want of fit in the distributions given in Table XXVII. can be ascribed to infection. This suggestion of an indeterminate period for incubation seems hardly reasonable, and has, so far as I know, in this class of disease no parallel. The "want of fit" in these distributions is possibly due to varying attack-rates on different mine properties, combined with irregularities due to the fact that all the Natives in these groups had not been under observation for precisely the same period, and also that the attack-rate varies with the period of residence. The improvement in the "fit" of the distribution shown in Table XXXI. when these two latter factors are allowed for is, as we have seen, very marked.

Before leaving this question of the infectiousness of pneumonia there is one other point to which attention may be drawn. We have already shown that the attack-rate of pneumonia is greatest on the arrival of Natives on the Rand, and gradually decreases as the period of residence increases (see Tables III. and IV.). The usual history of an outbreak of an infectious disease is of an entirely different character; the attack-rate as recorded day by day rises more or less rapidly for some days until the maximum is reached, when a steady, but somewhat slower, decline takes place. In the case of pneumonia amongst "Tropical Natives" we find the onset to be explosive in character, the attack-rate being at its maximum in the first twenty-four hours after arrival on the Rand, and thereafter falling rapidly; so that a curve showing the attack-rate according to length of residence on the "gold fields" is of the "J" type (see Charts I. and II.); such a curve would, I think, be unique as representing the history of an outbreak of an infectious disease.

In conclusion, a study of these figures shows definitely that if infection from case to case play any part in determining the prevalence of this disease among "Tropical Natives," it can be, at most, but a very small one; and moreover we can obtain no evidence of its existence. On the other hand, pneumococcal coryzas would appear, at least among Europeans, to be markedly infectious. The way in which catarrhal attacks often apparently associated with the pneumococcus, seem to spread from person to person throughout a household, or among the members of an office, strongly suggest case to case infection, nevertheless no such phenomena is apparent in the case of pneumonia. Nor, among these Natives, is pneumonia commonly preceded by catarrhal conditions of the naso-pharynx, or diseases of the influenzal type.

Finally, attention may be again drawn to the fact that this section deals only with the evidence in regard to infection from case to case among the Natives themselves. It has been suggested that the outbreak of pneumonia among these Natives is due to their infection with the pneumococcus contracted from Europeans with whom they associate when travelling to the mines. At present there is no direct evidence available in connection with this problem; it is one which will require investigation by other methods.

SECTION IV.

A consideration of The Effect of Prophylactic Inoculation of a Pneumococcal Vaccine on the Incidence and Mortality of Pneumonia.

A brief account only of the history of the preventive inoculation for pneumonia on the Rand need be given here. Full details of the bacteriological aspect of the investigation will no doubt be found in Sir Almroth Wright's expected report, and I shall confine myself to considering the results of some of the experiments from the statistical point of view.

Sir Almroth Wright, on the invitation of the Governing body of the Witwatersrand Native Labour Association, arrived in Johannesburg at the end of September, 1911. The first prophylactic inoculations were made on October 4th, in the W.N.L.A. compound; "Tropical Natives" only were treated. The method adopted was, first, to exclude from a gang any sick Natives, or those who appeared to be sickening for some illness, and then to divide the remainder into halves: the members of one half received a prophylactic inoculation, those of the other half being kept under observation as controls. These inoculations were made as soon as possible after the arrival of the Natives in the compound, and thus they were under the direct observation of Sir Almroth Wright and his assistants, for a period of about three weeks before being sent to work on the mines.

The first two gangs treated received a dose of vaccine of forty and sixty millions respectively; this vaccine was prepared in London. Subsequently a locally prepared vaccine was used, the doses being increased to 100, 150, and 200 millions. All inoculated Natives received two doses of vaccine unless they fell sick before the second was due. The interval between the injections varied from about seven to twelve days. In February the dose was increased to 300 millions. This series of inoculations I shall refer to as the "*first experiment.*"

While these inoculations were being carried out in the W.N.L.A. Compound large numbers of Natives were being similarly treated in some of the mine compounds along the Reef. This procedure has unfortunately made it impossible to satisfactorily follow the history of the experimental Natives leaving the W.N.L.A. Compound, for it was discovered that some of the controls were being inadvertently inoculated, and that inoculated Natives were being reinoculated, on the mines. The inoculation of the controls after a period of residence on the Rand, and their transference from the group "control" to the group "inoculated" constituted a very serious source of error. This will be at once apparent when we recollect that the attack-rate falls rapidly with the increase in the period of residence; and consequently, even if the inoculation itself were of no value, such a transfer at any date subsequent to the commencement of the observations would give rise to an apparent correlation in favour of inoculation. As it appeared impossible to satisfactorily estimate the magnitude of this source of error, I have confined myself to dealing with the results of this investigation only in so far as they were observed in the W.N.L.A. Compound.

At the commencement of the following recruiting season for "Tropical Natives"—August, 1912—a further experiment was planned, the preparation of the vaccine, and the inoculation of the "boys," being conducted by Dr. Parry Morgan, one of Sir Almroth Wright's assistants. Approximately 8,000 Natives were dealt with, and I shall refer to this investigation as the "*8000 experiment*." In this experiment again all "Tropical Natives" on arrival in the compound were divided into three groups—sick Natives were excluded, and given the distinguishing letter "F," and the remainder were divided into two classes by the inoculation of four and the omission of the fifth, who was regarded as a control and distinguished by the letter "E," the inoculated Natives being also given distinguishing letters according to the dose of vaccine they received. Every Native in this experiment was traced for a period of at least six months. After allotment, returns were received from the mines when any of these Natives were admitted to hospital, died, or were repatriated, together with the diagnosis made by the Medical Officer of the mine. I believe these returns are as exact as it is possible to get them, and they may be safely regarded as closely approximating to the real conditions. The doses of vaccine inoculated varied from 250 to 2,500 millions. A few much larger doses were also tried, mainly to determine if very large doses would give rise to toxic symptoms. The method of preparing the vaccine also varied, but it can be divided into two main classes; vaccine in which glucose was added to the medium in which the organism was grown, and that in which it was omitted. The main ingredients of the medium were broth with blood-serum and hæmoglobin, to which in some cases glucose was added. These two vaccines will be subsequently referred to as "Blood Vaccine" and "Glucose Vaccine."

The following table shows the nature of the vaccine, the dose, and the number of cases, in each group of this experiment.

TABLE XXXIII.

"8000 Experiment."
Results over period of six months.

					PNEUMONIA.			
Letter.	Nature of Vaccine.	Dose in Millions.	Number in Group.	Mean pop. for six months.	Cases.	Attack-rate per thousand.	Deaths	Case mortality per cent.
B	Blood	250	646	613	81	132	25	31
C	,,	500	759	722	90	124	26	29
A+C	,,	600°	162	158	17	108	2	12
D+H	Glucose	1250*	157	154	22	143	3	14
G	,,	500	463	440	46	104	19	41
H	,,	1000	650	627	69	110	17	25
J	,,	2500†	1661	1572	187	119	55	29
K	Blood	1500‡	1651	1564	175	112	55	31
Q	Various	Odd 200 —40000	75	72	7	97	3	43
E	Controls	0	1545	1439	231	160	74	32
F	Omitted from experiment	0	354					

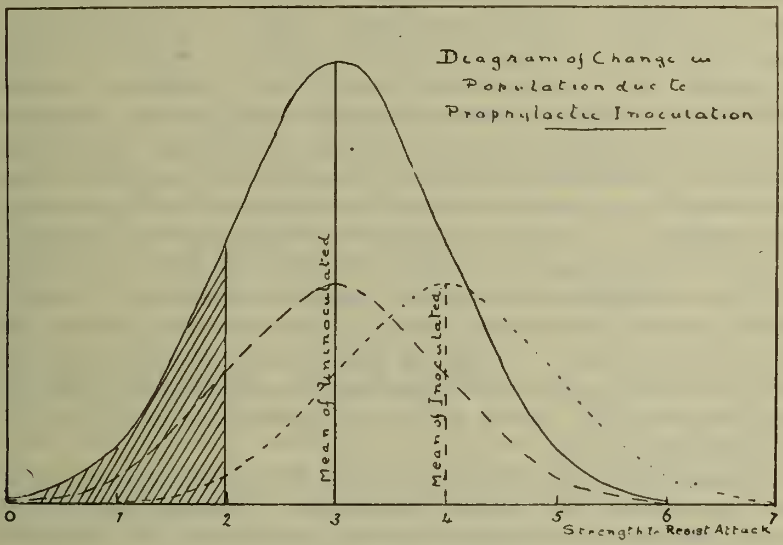
° First dose 100, second 500.
* First dose 250, second 1,000.
† Doses varied from 2,400 to 3,000, majority 2,500.
‡ Doses varied from 1,250 to 1,875, majority 1,500.

A general inspection of this table shows, first, that the attack-rate was highest amongst the controls, but that the case mortality in this group is not higher than that of several of the inoculated groups. Owing, however, to the small numbers comprised in many of these groups errors due to sampling will be considerable, and to estimate properly the effect of vaccination we must adopt other methods than those of mere inspection of the figures and rates. The effect of inoculation can be studied in relation to both the attack-rate and the death-rate from pneumonia. Any modification of the severity of the attack can be measured either by the case mortality, or by the length of the illness, if it were possible to obtain uniform data in regard to this latter point. The period of stay in the mine hospital was stated in the returns received from the mines, but on enquiry it became evident that this information would not be of any value for the purpose of determining the severity of the attack, because in many mine hospitals no case of pneumonia is discharged under a minimum period. Any results reached by considering the period spent in hospital (as regards the returns from the mines) will, for this reason, be unsatisfactory.

We have, therefore, three measures by which we can estimate the effect of prophylactic inoculation against pneumonia; namely, (1) the attack-rate, (2) the death-rate, and (3) the case mortality from this disease. The question arises as to what statistical methods we shall employ in order to obtain the best guide to the results of this procedure. One naturally turns first of all to the "four-fold table"[25] method of Professor Pearson, the tetrachoric r, (r_t) as it is now called. This method has been much used for similar material in relation to vaccination against smallpox, anti-typhoid inoculation, and other enquiries of the same nature. Recently, however, Mr. G. U. Yule has attacked the use of this method for dealing with such problems,[15] and has suggested as preferable the employment of certain "coefficients of association." A full reply to these criticisms of Mr. Yule, and a demonstration that his suggested measures are unsuitable for problems such as those before us, has been made by Professor Pearson and Dr. Heron.[16] The objection to the tetrachoric r is that this method assumes that the distribution of the variates is continuous, and that they are Gaussian in type. It has, however, been shown that a very considerable departure from normality does not seriously affect the results obtained by this method, provided that the dichotomies are not taken too near the tails of the distribution. The nature of our distributions is unfortunately such, that the divisions in many instances occur in such a situation that only small frequencies are obtained in two of the cells. These inevitably small frequencies constitute an objection to the use of this method for solving these problems, because we are unable to *assert* that the distribution of our variates is approximately Gaussian in type. Mr. Yule would, apparently, even consider them discontinuous; for as he says, A man is either "dead" or "not dead"; and presumably also either "attacked" or "not attacked" by any specific disease. This is a question of fundamental importance; and although it has been fully answered by Pearson and Heron in the paper just referred to, and was also dealt with by Greenwood at the time of its enunciation,[17] yet it would appear desirable to deal briefly with it here; for it is apparently one which presents difficulties to medical readers, to whom the literature referred to may not always be readily accessible.

Let us consider the problem in regard to recoveries and deaths in a population of those attacked by a disease. We wish to measure the virulence of an outbreak of the disease by the ratio of recoveries to deaths. Are we then to consider the distribution continuous or discrete? If we group our data into "dead" and "not dead," and consider the groups as discrete, and differing only by the presence or absence of a character (life), we are assuming that the disease can be best represented on the supposition that it has only two grades of virulence. If on the other hand we regard virulence as a continuous variate we shall allow for every grade of severity in attack, and consider the dividing line (death) merely to cut this distribution at some particular place. The fact that the continuous distribution is assumed to continue after the position of the dichotomy appears to constitute a difficulty to some. The medical reader will perhaps more easily appreciate the conception of continuity by means of a simple illustration. Let us imagine a number of pellets, shot from a fixed air gun, to travel exactly in the same direction, they will not, nevertheless, all come to rest at the same spot; but their positions can when enough have been discharged be represented by some continuous curve. If they were allowed to fall upon a tray divided into compartments we should probably find that in the first compartment a stray pellet or so was present, the number in subsequent compartments increasing regularly up to a certain compartment and thereafter decreasing. That is to say, we must consider the velocity of the pellets as a continuous function, influenced it may be by many factors. If we introduce a sheet of paper at any position on the range so that some of the pellets fall short of the paper whilst others pierce it, it would not now be justifiable—in fact it would be quite misleading—to consider that our distribution could be better considered as discrete and divisible into two classes—those that " pierce " and those that do " not pierce " the paper. Clinical observation teaches us that we have in most diseases a range of all degrees of severity, from mere indisposition up to death, and to assume that a break in the continuity of the range occurs at death, or that the range terminates abruptly at this point is, I think, unscientific, particularly as experience shows that while some people appear only just to fail to overcome the disease, others have seemingly from the commencement no hope of recovery. It has been shown that in some diseases at any rate, the normal curve may as a first approximation be used satisfactorily to describe such distributions. Even if a Type I. curve, such as those shown in Charts VII. and VIII., is a more usual form than the Gaussian, very little error will be introduced in the value of r_t by assuming the distribution to be normal, unless the dichotomies are made at the extreme limits of the range.

It seems to me that the " four-fold " table method would therefore be quite satisfactory for ordinary use in regard to the variate " recovery and death," or " attacked and not attacked." But in regard to the other variate, more difficulty arises: are we to consider " inoculated and uninoculated " as represented by a continuous distribution of more or less Gaussian type? We desire to measure the protection conferred by inoculation. My conception of the problem is as follows: Members of a population possess the character "power to resist attack" in varying amounts, and the distribution of this population in regard to the character is continuous and probably capable of being represented, at least approximately, by a normal curve. Assuming inoculation to increase this "power to resist attack," its effect will be to bodily move the treated portion of this population in the direction of increasing amounts of this power, without, necessarily, altering the type of the distribution.



The accompanying diagram illustrates this suggestion. The continuous curve represents the original population in respect to the factor "power to resist attack." The broken curves are supposed to represent the population after one half has received a prophylactic dose of some vaccine. The curve representing the inoculated population has been moved to the right, but is otherwise unaltered. The shaded portion of the diagram, ending at the ordinate "2," is supposed to represent diagramatically the portion of the population who are attacked. It will be seen that the effect of shifting the curve representing the inoculated population to the right, is to reduce the area which falls in the shaded portion of the diagram, i.e., to reduce the number of the population who contract the disease.

If this conception of the effect of inoculation be correct, the amount of shift in the position of the mean would give us a measure of the protection conferred by inoculation. If the distributions were truly Gaussian the displacement in the mean of the inoculated population could easily be determined from a table of the "Probability Integral" when the attack-rates were known. But unfortunately we cannot *assert* that the distributions are Gaussian, and large errors may arise when the attack or death rates are small, if the distribution should not be so. Further, when only small areas at the tail are relied on for calculating the position of the mean errors of random sampling, unless very large total numbers are being dealt with, may introduce serious errors.

Again, the two sub-distributions might be considered as concentrated at their means, and then regarded as differing by a unit "something" from each other, with a mean equidistant from each group when so concentrated. If we equalise the groups, we could by using the "New Correlation Method" described by Professor Pearson,[23] rapidly calculate the correlation. This method of equalising the sub-groups is arbitrary, and has not, I think, any theoretical justification; nevertheless, it has the advantage of enabling an approximate value of the correlation to be found in a few moments if a copy of Sheppard's "Tables of the Probability Integral"[26] be at hand. In a few of the following tables the value of "r" thus obtained has been added (under the symbol r_h); it will be noticed that this value rarely differs by more than the probable error from the value of r_t. So close an agreement in the two values does not always occur, and in a few experimental examples where the value of r_t was large the values differed by considerably more than the probable error.

Considering these difficulties, which I have discussed at some length because of their intrinsic importance in medical problems, we cannot consider the "four-fold" method as entirely satisfactory for our present purpose, nevertheless, if a correlation value is desired it is, I believe, the best method at present available for this purpose. Fortunately, however, we can also obtain a direct measure of the improbability that our observed distributions have arisen by errors of random sampling, without making any supposition as to the nature of the distribution, or even as to whether the variates are continuous or discrete. To determine this value the "goodness of fit"[14] is calculated, the value of P for given χ^2 for $n'=4$ is rapidly obtained from Elderton's Tables.[22] When, however, P is very small, it is impossible to grasp its significance, a difficulty which has been met by Professor Pearson by the introduction of a *correlation scale of the improbability of independence*.[18] This constant is denoted by the symbol η_p

In the analysis of the following tables I have calculated the tetrachoric "r" (r_t) because, although not perfect for this purpose it is useful as a control, and is also useful for purpose of comparison with similar problems in which it has previously been employed. Everett's Tables of Tetrachoric Functions[24] were used, and the probable error calculated by the method given by Pearson.[21] The probability of the variations obtained having arisen by chance was also calculated by the method of "goodness of fit," and is given in each case, and in addition, in some instances, the value of "r_p" as mentioned above, ${}_0\sigma_r$ being determined by the aid of Professor Pearson's tables, and r_t then obtained directly from Mr. Soper's Abac.[18] In the calculation of χ^2 the theoretical values were taken to the nearest unit.

Before turning to the actual results obtained by these methods, it will be well to consider briefly the meaning of the word "significant" when applied to a correlation coefficient; that is to say, the interpretation of a result in relation to its probable error. It is no doubt a matter of opinion, to a certain extent, as to what degree of probability we should demand before a value can rightly be considered as significant. No reasonable person would consider, that a variation in a distribution which is as likely as not to arise owing to errors of random sampling was significant evidence of the effective introduction of any new factor in the causation of the variation. If, however, the odds against a certain distribution arising by chance are, let us say, four to one, can we then consider such a result as significant of the introduction of a new factor? Clearly not! although such a result might be considered as indicating a certain "a priori" reason for repeating the experiment, yet no deductions as to its probable influence could be legitimately drawn from such a result. The odds which we should demand before considering a result as significant must be a matter for individual judgment, and those who have had most experience of this kind of analysis will be best able to form a reliable estimate of the significance, or otherwise, of any particular value. The usual practice amongst biometric workers is to consider a coefficient of correlation as probably significant if its value is greater than three times the value of its probable error. For general purposes this is probably a fairly safe measure, but it should be remembered that even a guide of this sort, although showing that the chances are certainly in favour of the observed correlation being significant, does not constitute a definite proof that it is so, and all collateral evidence must receive due weight. It is, however, in this sense that the term "significant" is used in this paper.

First, let us deal with the results of what we have termed the "first experiment." The data in regard to the number attacked with pneumonia are only available for the first few weeks after inoculation. The number attacked in each group, inoculated and uninoculated, is shown in this table.

TABLE XXXIV.

	Inoculation.		
	+	—	Total.
Not attacked	5822	5494	11316
Attacked	149	195	344
Total	5971	5689	11660

$r_t = \cdot 0876 \pm \cdot 0196$

$\chi^2 = 8\cdot 73917$ $P = \cdot 0337$

$r_p = \cdot 06$ $r_k = \cdot 07$

There is a small but appreciable correlation in favour of inoculation, as reducing the attack-rate; and the chances are 29 to 1 against so large a variation from the expected arising owing to errors of random sampling.

If we employ the number of deaths, instead of the number attacked, as the measure of the effect of the inoculation, we obtain the following results:—

TABLE XXXV.

	Inoculation.		
	+	—	Total.
Not dead	5922	5602	11524
Dead	49	87	136
Total	5971	5689	11660

$r_t = \cdot 1475 \pm \cdot 0268$

$\chi^2 = 13\cdot 13498$ $P = \cdot 0044$

$r_p = \cdot 13$ $r_k = \cdot 12$

Here the odds against such a variation from the expected as this arising by chance are 227 to 1. We may, therefore, regard both these correlations, although small, as significant.

A table of recoveries and deaths amongst the inoculated and uninoculated in this group can be constructed, and is as follows:—

TABLE XXXVI.

	Inoculation.		
	+	—	Total.
Recoveries	100	108	208
Deaths	49	87	136
Total	149	195	344

$r_t = \cdot 1909 \pm \cdot 0567$

$\chi^2 = 4\cdot 95219$ $P = \cdot 1761$

In this table the value of r_t is, according to our definition, just significant, and the odds against a variation as large as this arising by chance are only 4·7 to 1.

Finally, from the figures obtained from this experiment we can form a "three by two fold" table, as under:—

TABLE XXXVII.

	Inoculation.		
	+	—	Total.
Not attacked ...	5822	5494	11316
Attacked, recovered...	100	108	208
Attacked, died ...	49	87	136
Total	5971	5689	11660

$\chi^2 = 12{\cdot}96818 \quad P = {\cdot}0237$

The chances are, therefore, about 42 to 1 against this distribution having arisen owing to errors of random sampling.

Turning now to an examination of the data obtained from the "8000 experiment," the returns from which are available for a period of six months, we can construct the following tables. In these tables the *average* number of Natives exposed to risk for the period dealt with is used, not the number *actually* inoculated, because some of the Natives died from other causes and some were repatriated during the period of observation. Tables showing the number attacked and the number who died are given below.

TABLE XXXVIII.

	Inoculation.		
	+	—	Total.
Not attacked ...	5228	1208	6436
Attacked	694	231	925
Total	5922	1439	7361

$r_t = {\cdot}1132 \pm {\cdot}0180$

$\chi^2 = 19{\cdot}64246 \quad P = {\cdot}0002$

$r_p = {\cdot}10 \quad r_k = {\cdot}10$

TABLE XXXIX.

	Inoculation.		
	+	—	Total.
Not dead	5717	1365	7082
Dead	205	74	279
Total	5922	1439	7361

$r_t = {\cdot}1070 \pm {\cdot}0260$

$\chi^2 = 8{\cdot}49944 \quad P = {\cdot}0377$

$r_p = {\cdot}06 \quad r_k = {\cdot}09$

These results confirm those already found from the "first experiment," in that they show that whether we employ the attack-rate or the death-rate as the measure of the protection afforded by inoculation, a small, but definite correlation in favour of this process is observed in all four tables.

TABLE XL.

	Inoculation.		
	+	—	Total.
Recoveries	489	157	646
Deaths	205	74	279
Total	694	231	925

$r_t = \cdot 0424 \pm \cdot 0397$

$\chi^2 = 0\cdot 43750 \quad P = \cdot 9130$

In regard to the effect of inoculation on the case mortality the results here are clearly not significant. In a similar table constructed from the data of the " first experiment " we found a higher correlation, but as the value of P was ·1761, it is not therefore very surprising to find a lower value here; nevertheless, as the correlation was just " significant " in the former table a further examination was undertaken. It seemed possible that the explanation of the difference in values might lie in the fact that the returns for the "8000 experiment" dealt with a six-month period, whereas the former were only concerned with a mean period of approximately four weeks from the date of inoculation, and if the beneficial influence in this respect were only of temporary duration its effect might be masked in the latter table. To estimate the value of this suggestion the following table was constructed by including only the cases which arose in the W.N.L.A. compound during the first month after inoculation.

TABLE XLI.

Case Mortality.

	Inoculation.		
	+	—	Total.
Recoveries	105	32	137
Deaths	71	29	100
Total	176	61	237

$r_t = \cdot 1085 \pm \cdot 0742$

$\chi^2 = 0\cdot 81315 \quad P = \cdot 8384$

Here again, although the absolute value of the correlation is increased it cannot in relation to its probable error be considered as significant, and the value of P indicates that variations as great as this are to be expected in samples of this size. It seems, therefore, that while we can state that prophylactic inoculation has a value in diminishing the attack-rate from pneumonia, there is at present no definite evidence that it has much influence in lowering the case mortality. It is possible that larger numbers when available may definitely settle this point, but so far as the present returns go we have only a suggestion that a small reduction in the case mortality follows preventive inoculation, and this possibly is most marked for a short period following inoculation. In this connection one point remains to be noted; the large majority of Natives comprised in the inoculated group of the "first experiment" received two comparatively small doses of prophylactic vaccine, while those composing this group in the "8000 experiment," with a few exceptions, received only one dose. Whether this factor had any effect on the result further observation can alone definitely determine, the following considerations may, however, be of some interest.

A reference to Table XXXIII. shows that the two groups, in the "8000 experiment," which received two doses of prophylactic vaccine, viz.: (A + C) and (D + H) had a lower case mortality than any of the other groups; but the number of cases concerned is so small that no reliance can be attached to such results, and more especially so when we remember how very variable the case mortality from this disease is liable to be. It was, nevertheless, suggestive; and I have, therefore, tabled the records of 146 cases of pneumonia treated in the W.N.L.A. Hospital at the time of the "first experiment," and have grouped them according to whether they had received two or only one dose of prophylactic vaccine.

TABLE XLII.

	Inoculation.		
	+	++	Total.
Recovered	55	44	99
Died	18	29	47
Total	73	73	146

$r_t = \cdot 2631 \pm \cdot 0867$

$\chi^2 = 3\cdot 13883 \quad P = \cdot 3736$

This correlation, if considered as significant, is in favour of the cases which received only one prophylactic dose of vaccine. There are two possibly disturbing factors to be considered, (1) twenty-seven of the cases in the first group, as against twenty-two in the second, were *treated* with a pneumococcal vaccine; this, as we shall see later, would, if it had any effect, tend to reduce the correlation, and consequently the observed value would be too small: (2) the cases appearing in the first group occurred, on the average, sooner after arrival in the compound than those in the second group. This was due to the fact that, with very few exceptions, all these Natives received two inoculations, unless they had fallen sick in the interval after the first dose and before the second was due. So that cases among the inoculated occurring within a week or ten days of arrival will appear in the first group and subsequent cases in the second group. We must recollect, however, that the case mortality does not appear to be a function of the period of residence on the Reef; and if this factor has any influence, it may be due to the fact that the cases in the first group occur on the whole with a shorter interval between inoculation and attack than those in the group with two prophylactic inoculations. Although, as previously stated, the majority of Natives comprising the "first experiment" received two doses of prophylactic vaccine, and those in the "8000 experiment" only one, yet no satisfactory conclusions in respect to the relative value of one or two doses can be drawn from the case mortality rates in the two groups. First, because many of the attacked in the earlier part of the period —the only portion for which we have reliable records—had, as just explained, only received the first dose; secondly, the season during which they were under observation was not the same, and the case mortality varies considerably from time to time; thirdly, the doses of the vaccine given are not comparable in the two experiments.

The "three by two fold" table for the "8000 experiment." corresponding to that given for the "first experiment," is as follows:—

TABLE XLIII.

	Inoculation.		
	+	—	Total.
Not attacked ...	5228	1208	6436
Recovered	489	157	646
Died	205	74	279
Total	5922	1439	7361

$\chi^2 = 20{\cdot}120392 \quad P = {\cdot}0011$

Thus only once in a thousand samples should we expect a result as bad as this if the variations were due to errors of sampling.

Having seen that a certain amount of protection against attack is conferred by inoculation, we must proceed to investigate the period for which such protection lasts. Table XLIV. gives the monthly details in regard to the number attacked, and the number who died (with the rates) among the inoculated and controls comprised in the "8000 experiment."

This table was constructed by placing in the group 1st month all cases of pneumonia which occurred within thirty days of the date of inoculation, and so on; and, the deaths in each group refer to deaths among the attacked in each month after inoculation. Thus although a Native may have died on the fortieth day from the date of inoculation he will appear as a death in the first month if the disease was contracted within thirty days from inoculation.

TABLE XLIV. A.

Column 0 = Month after arrival, 1 = Average population, 2 = Pneumonia cases, 3 = Pneumonia deaths, 4 = Other respiratory diseases cases, 5 = Other respiratory diseases deaths, 6 = Other medical diseases cases, 7 = Other medical diseases deaths, 8 = Repatriations.

INOCULATED.									UNINOCULATED (CONTROLS).							
0	1	2	3	4	5	6	7	8	1	2	3	4	5	6	7	8
1st	6170	179	71	109	2	273	38	2	1523	68	29	42	2	70	12	1
2nd	6079	140	34	194	3	369	13	18	1488	52	15	48	1	94	1	11
3rd	6000	116	23	185	2	362	6	60	1459	33	8	45	0	105	2	19
4th	5882	100	33	138	1	276	11	98	1425	34	9	39	1	65	1	29
5th	5757	90	22	102	5	235	8	72	1386	28	5	37	0	56	1	31
6th	5648	69	22	114	2	187	10	76	1356	16	8	37	1	45	2	15
Total	5922	694	205	842	15	1702	86	326	1456	231	74	248	5	435	19	106
7th	2556	36	12		3		2	34	649	9	5		1		1	11
8th	2518	31	6		0		0	20	637	7	0		0		1	6
9th	2492	20	7		1		4	13	628	8	4		1		0	6

TABLE XLIV. B.

Rates.

Column 0=Month after arrival, 9=Pneumonia attack-rate per thousand, 10=Pneumonia death-rate per thousand, 11=Pneumonia case-mortality per cent., 12= Respiratory diseases death-rate per thousand, 13=Respiratory diseases case-mortality per cent., 14= Other medical diseases death-rate per thousand, 16=Other medical diseases case-mortality per cent., 16=Repatriations per thousand.

	INOCULATED.								UNINOCULATED (CONTROLS).							
0	9	10	11	12	13	14	15	16	9	10	11	12	13	14	15	16
1st	29·0	11·5	39·7	0·32	1·8	6·16	13·9	0·32	44·6	19·0	42·6	1·31	4·8	7·88	17·1	0·66
2nd	23·0	5·6	24·3	0·49	1·5	2·14	3·5	3·0	34·9	10·1	28·8	0·67	2·1	0·67	1·1	7·4
3rd	19·3	3·8	19·8	0·33	1·1	1·00	1·7	10·0	22·6	5·5	24·2			1·37	1·9	13·0
4th	17·0	5·6	33·0	0·17	0·7	1·87	4·0	16·7	23·9	6·3	26·5	0·70	2·6	0·70	1·5	20·4
5th	15·6	3·8	24·4	0·87	4·9	1·39	3·4	12·5	20·2	3·6	17·9			0·72	1·8	22·4
6th	12·2	3·9	31·9	0·35	1·8	1·77	5·4	13·5	11·8	5·9	50·0	0·74	2·7	1·47	4·4	11·1
Av. monthly rate	19·5	5·8		0·42		2·4		9·2	26·4	8·4		0·57		2·20		12·2
7th	14·1	4·7	33·3	1·17		0·78		13·3	13·9	7·7	55·6	1·54		1·54		16·9
8th	12·3	2·4	19·4					7·9	11·0					1·57		9·4
9th	8·0	2·8	35·0	0·40		1·61		5·2	12·7	6·4	50·0	1·59				9·6

An inspection of this table shows that for the first two months the attack-rate amongst the inoculated is considerably lower than amongst the controls. Four-fold tables for the attack-rate for each month have been prepared, and are as follow:—

TABLE XLV.

A—1st month.

	Inoculation.			
	+	—	Total.	
Not attacked ...	5991	1455	7446	$r_t = \cdot1138 \pm \cdot0268$
Attacked	179	68	247	$\chi^2 = 9\cdot49594 \quad P = \cdot0221$
Total	6170	1523	7693	

B—2nd month.

	Inoculation.			
	+	—	Total.	
Not attacked ...	5939	1436	7375	$r_t = \cdot1053 \pm \cdot0296$
Attacked	140	52	192	$\chi^2 = 6\cdot56665 \quad P = \cdot0891$
Total	6079	1488	7567	

C—3rd month.

	Inoculation.			
	+	—	Total.	
Not attacked ...	5884	1426	7310	$r_t = \cdot0373 \pm \cdot0325$
Attacked	116	33	149	$\chi^2 = 0\cdot69897 \quad P = \cdot8611$
Total	6000	1459	7459	

D—4th month.

	Inoculation.			
	+	—	Total.	
Not attacked ...	5782	1391	7173	$r_t = \cdot0808 \pm \cdot0332$
Attacked	100	34	134	$\chi^2 = 3\cdot11217 \quad P = \cdot3770$
Total	5882	1425	7307	

E—5th month.

	Inoculation.			
	+	—	Total.	
Not attacked ...	5667	1358	7025	$r_t = \cdot0596 \pm \cdot0357$
Attacked	90	28	118	$\chi^2 = 1\cdot37287 \quad P = \cdot7159$
Total	5757	1386	7143	

F—6th month.

	Inoculation. +	Inoculation. —	Total.	
Not attacked ...	5579	1340	6919	$r_t = -\cdot 0077 \pm \cdot 0406$
Attacked	69	16	85	$\chi^2 = 0\cdot 00000$ $P = 1\cdot 00000$
Total	5648	1356	7004	

These tables show that after the first two months there is very little evidence in favour of the protective influence of this vaccination, but as the number of the controls selected was small, variations in rates due to small samples is considerable. The variation in rates is shown graphically in Chart XIa., where it will be noticed how much more smoothly the rates run for the inoculated than for the uninoculated; the former are based on approximately four times the number of cases. Returning to the actual figures we notice that for the 6th month, and so far as the figures are available for the 7th and 8th months, the attack-rates among the uninoculated are actually a little less than among the inoculated. These differences are clearly not significant, and are again reversed in the 9th month. We can, I think, sum up this part of the evidence by saying that these returns indicate that any protection obtained lasts for only a limited period after inoculation, and that the general trend of the figures suggests that it does not entirely disappear until about the fifth month, although there is but little positive evidence that much of its efficacy remains after the second month. This falling off is even more marked when we exclude second attacks from the returns, as is shown in Chart XIb.

The prevalence of second attacks of pneumonia occurring among the inoculated and the controls is of some interest. We have already seen—on the basis of our previous definition of "second attacks"—that one attack does not apparently confer any immunity against a second. From the figures given below we see that a higher value is obtained for the correlation for immunity against second attacks amongst the inocluated, than that for the general attack-rate.

TABLE XLVI.

	Inoculation. +	Inoculation. —	Total.	
First attack only ...	514	146	660	$r_t = \cdot 2655 \pm \cdot 0532$
Second attack ...	50	33	83	$\chi^2 = 12\cdot 5328$ $P = \cdot 0061$
				$r_p = \cdot 23$ $r_k = \cdot 22$
Total recoveries from first attack ...	564	179	743	

TABLE XLVII.

	Inoculation.			
	+	—	Total.	
Recovery 2nd attack...	37	26	63	$r_t = -\cdot0955 \pm \cdot1283$
Death in ,, ...	13	7	20	$\chi^2 = 0\cdot27169 \quad P = \cdot9460$
Total	50	33	83	

The table for the attacks is constructed from all recoveries from first attacks, and is not limited to a six-month period. As the period of exposure to risk of a second attack varied the calculation of rates, except for the comparison of the one group with the other, will be without value. Although the second attack-rate amongst the inoculated appears to be considerably reduced there is no evidence that the case mortality is favourably affected. An examination of the length of the interval between the first and second attacks would seem to suggest that the diminution in the rate among the inoculated is chiefly confined to a period of thirty days after the primary attack. The figures when thus sub-divided are very small, and consequently but little weight can be attached to this observation unless confirmed by future records.

The question of the effect of the variations in the size of the dose of protective vaccine can now be considered. The inoculated Natives are divided into two groups, (1) those receiving medium doses, viz., the sub-groups B, C, (A + C), G, H, and (H + C), and (2) those classed as large doses, viz., sub-groups J, K, and Q. These two groups, even when the question of dose is excepted, are not strictly comparable, owing to the fact that their arrival on the Rand did not take place at the same time of the year, and the attack-rate shows, as we have seen, a seasonal variation. As a check on these results it is advisable, therefore, to divide the controls into two groups, to correspond with those for the inoculated.

TABLE XLVIII.

	Inoculated (size of dose).			
	Large.	Medium.	Total.	
Not attacked ...	2841	2387	5228	$r_t = \cdot0153 \pm \cdot0180$
Attacked	369	325	694	$\chi^2 = 0\cdot42096 \quad P = \cdot9163$
Total	3210	2712	5922	

TABLE XLIX.

	Controls to Large.	Medium.	Total.
Not attacked ...	629	579	1208
Attacked	106	125	231
Total	735	704	1439

$r_t = {\cdot}0857 \pm {\cdot}0334$

$\chi^2 = 2{\cdot}97172 \quad P = {\cdot}3967$

Neither of these results can be considered as really significant, but any evidence in favour of the group "large dose" is more marked among the controls than the inoculated.

If the material be grouped according to the size of the dose—taking 250 millions as the unit for the group—the following table can be constructed from which a correlation value can be found by the new method described by Professor Pearson.[23]

TABLE L.

Dose of vaccine ...	250	500	750	1000	1250	1500	1750	2000	2500	Total.
Not attacked ...	532	1167		558	132	1391			1386	5166
Attacked	81	153		69	22	175			187	687
Total	613	1320		627	154	1566			1573	5853

$r = -{\cdot}0164 \pm {\cdot}0157$* $\quad \chi^2 = 3{\cdot}27686 \quad P = {\cdot}9850$

In this table the Natives comprising the group "Q" are omitted. We again obtain what is practically a zero correlation between the size of dose and freedom from attack. The rates are naturally above the mean in the groups 250, 1,250, and 2,500, and below it in groups 500, 1,000, and 1,500; while the variations are not greater than, or even so great as, might be expected to arise from errors of random sampling. The case mortality is also, so far as these figures go, uninfluenced by the size of the dose, thus:—

TABLE LI.

	Inoculated (size of dose). Large.	Medium.	Total.
Recoveries	256	233	489
Deaths	113	92	205
Total	369	325	694

$r_t = -{\cdot}0423 \pm {\cdot}0424$

$\chi^2 = 0{\cdot}45766 \quad P = {\cdot}9090$

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* The probable error has been assumed to be $2\left(.67449\,\frac{(1-r^2)}{\sqrt{N}}\right)$

TABLE LII.

	Controls to			
	Large.	Medium.	Total.	
Recoveries	72	85	157	$r_t = -\cdot 0013 \pm \cdot 0727$
Deaths	34	40	74	$\chi^2 = 0\cdot 00000$ $P = 1\cdot 0000$
Total	106	125	231	

It has already been pointed out that the method of preparing the vaccine was varied by the addition, in some cases, of glucose to the medium in which the organism was grown. The returns can be analysed in respect of this point as follows:—

TABLE LIII. (*attack-rate*).

	Variety of vaccine.			
	Blood.	Glucose.	Total.	
Not attacked ...	2696	2469	5165	$r_t = -\cdot 0096 \pm \cdot 0181$
Attacked	363	324	687	$\chi^2 = 0\cdot 10577$ $P = \cdot 9790$
Total	3059	2793	5852	

TABLE LIV. (*case mortality*).

	Variety of vaccine.			
	Blood.	Glucose.	Total.	
Recoveries	255	230	485	$r_t = -\cdot 0133 \pm \cdot 0427$
Deaths	108	94	202	$\chi^2 = 0\cdot 02815$ $P = \cdot 9944$
Total	363	324	687	

The "Q" group is omitted from these tables as mixed vaccines were given to its members. In both these tables the correlation is virtually zero, and there is, therefore, no evidence that the addition of glucose to the culture media had any effect on the results.

The morbidity and mortality returns obtained in regard to the Natives constituting the "8000 experiment" included other diseases as well as pneumonia, we can, therefore, proceed to consider the influence of the prophylactic inoculation in relation to "other medical diseases."

Taking first the whole group "all medical diseases other than pneumonia," the morbidity table for the six months is as follows:—

TABLE LV.

	Inoculation.			
	+	—	Total.	
Not attacked	3378	761	4139	$r_t =$ ·0598 ± ·0142
Attacked	2544	678	3222	$\chi^2 =$ 8·08589 $P =$ ·0446
Total	5922	1439	7361	

In all the morbidity tables throughout this memoir it must be remembered that the number of attacked does not represent the number of *individual patients* admitted to hospital but the number of cases admitted; the same "boy" may have been, and, as a matter of fact was, particularly in regard to "other medical diseases," frequently readmitted on one or more occasions. It will be noted that a small but significant correlation in favour of inoculation is obtained from this group in relation to the attack-rate, but a zero correlation is obtained from the following mortality table.

TABLE LVI.

	Inoculation.			
	+	—	Total.	
Not dead	5821	1415	7236	$r_t =$ —·0052 ± ·0348
Dead	101	24	125	$\chi^2 =$ 0·00000 $P =$ 1·0000
Total	5922	1439	7361	

These returns can be further sub-divided into respiratory and non-respiratory diseases. Under the heading respiratory diseases, cases reported as colds, bronchial catarrh, broncho-pneumonia, coughs, influenza, tuberculosis, etc., are classified.†

The morbidity and mortality tables for Respiratory diseases, excluding Lobar Pneumonia, are as under:—

TABLE LVII.

	Inoculation.			
	+	—	Total.	
Not attacked	5080	1196	6276	$r_t =$ ·0657 ± ·0173
Attacked	842	243	1085	$\chi^2 =$ 6·60737 $P =$ ·0875
Total	5922	1439	7361	

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† Pulmonary tuberculosis accounted for in all sixty-two cases, fifteen of which died. Three-quarters of the deaths from Respiratory diseases other than pneumonia were, therefore, attributable to tuberculosis.

TABLE LVIII.

	Inoculation.			
	+	—	Total.	
Not dead	5907	1434	7341	$r_t =$ ·0645 ± ·0706
Dead	15	5	20	$\chi^2 =$ 0·32116 $P =$ ·9362
Total	5922	1439	7361	

Here again we obtain a correlation in favour of the inoculated in regard to the attack-rate from these diesases, but an insignificant value in regard to the death-rate. It will be observed that the case mortality in this group of diseases is low, less than 2 per cent. The number of deaths dealt with is so small that no conclusions of value can be drawn from this table. On sub-dividing the morbidity returns into (1) cases which occurred in the W.N.L.A. compound, i.e., early cases, and (2) cases reported from the mines, a drop in the value of the correlation corresponding to the lapse of time after inoculation is observed, a phenomenon similar to that found in the tables constructed from the pneumonia rates. The following are tables showing attack-rate from Respiratory diseases excluding pneumonia:—

TABLE LIX.

(Cases occurring in the W.N.L.A. compound.)

	Inoculation.			
	+	—	Total.	
Not attacked ...	5876	1418	7294	$r_t =$ ·1503 ± ·0432
Attacked	46	21	67	$\chi^2 =$ 6·17045 $P =$ ·1048
Total	5922	1439	7361	

TABLE LX.

(Cases occurring on the mines.)

	Inoculation.			
	+	—	Total.	
Not attacked ...	5158	1230	6388	$r_t =$ ·0425 ± ·0179
Attacked	764	209	973	$\chi^2 =$ 2·72033 $P =$ ·4422
Total	5922	1439	7361	

Very little weight can be attached to these values, nevertheless, so far as they go, they indicate a fall in the value of the correlation with the increase of time after inoculation.

The group "other medical diseases, excluding pneumonia," and "other respiratory diseases," yield zero correlations, and the probability is that the observed divergencies from the most probable distributions are merely due to errors of random sampling.

The following tables show the morbidity and mortality returns from other medical diseases excluding pneumonia and respiratory diseases.

TABLE LXI.

	Inoculation.			
	+	—	Total.	
Not attacked ...	4220	1004	5224	$r_t = \cdot 0248 \pm \cdot 0150$
Attacked	1702	435	2137	$\chi^2 = 1{\cdot}21132 \quad P = \cdot 7529$
Total	5922	1439	7361	

TABLE LXII.

	Inoculation.			
	+	—	Total.	
Not dead	5836	1420	7256	$r_t = -\cdot 0233 \pm \cdot 0369$
Dead	86	19	105	$\chi^2 = 0{\cdot}06264 \quad P = \cdot 9860$
Total	5922	1439	7361	

It would appear then that while prophylactic inoculation has apparently some influence in lowering the attack-rate from respiratory diseases other than lobar pneumonia there is no evidence that it has any influence in respect to non-respiratory diseases. This influence on respiratory diseases may be a measure of the prevalence of pneumococcal infections in this group. We know that in catarrhal conditions of the naso-pharynx the pneumococcus can frequently be recovered from the secretions in almost pure culture. In some cases of broncho-pneumonia it can be obtained by lung-puncture, and may also be found in many cases in the sputum. It is of interest in this connection to record that Dr. Watkins-Pitchford informs me that during the last year the pneumococcus has been obtained in practically pure culture from nearly all cases of so-called "Influenza" examined in the routine laboratories of the Institute, and that the influenza bacillus has been but rarely seen; in previous years, however, the influenza bacillus was frequently recovered from such cases.

Summarising the results obtained by this analysis of the experimental prophylactic inoculation with pneumococcal vaccine, we find:

(1) That the attack-rate from pneumonia is apparently lessened by inoculation, a small positive correlation being obtained.

(2) That the beneficial results observed are most marked during the first month after inoculation, and that they gradually diminish until at about the end of the fourth month, no protective power apparently remains.

(3) That there is little or no evidence that the case mortality is favourably affected by inoculation. Possibly the fatality of the disease may be reduced for a short period after inoculation.

(4) That there is no evidence to indicate that there is an optimum dose of vaccine to procure the maximum protection, nor whether two prophylactic injections are of more value than a single dose.

(5) That the addition of glucose to the medium for the preparation of the vaccine is associated with no apparent improvement in the results.

(6) That the attack-rate from other respiratory diseases is also favourably influenced by this vaccine; this is probably due to the fact that many of these diseases are causally associated with the pneumococcus.

(7) That other medical diseases are uninfluenced by the prophylactic use of this vaccine.

In addition to organising the prophylactic inoculations against pneumonia Sir Almroth Wright conducted a small number of experiments in regard to the curative value of this vaccine. Alternate cases of pneumonia, occurring amongst the previously vaccinated and the unvaccinated Natives admitted to the W.N.L.A. Hospital, received treatment with vaccine. The hospital case-sheets of the patients thus selected for treatment, as well as those of the controls, were appropriately marked. I have been able to trace 307 such sheets, of which 146 were marked "treated" and 161 "not treated." The correlation obtained between vaccine treatment and recovery is negative in sign, but having regard to its probable error, it is not significant.

TABLE LXIII.

	+	—	Total.	
	Vaccine treatment.			
Recoveries	93	112	205	$r_t = -\cdot 1099 \pm \cdot 0608$
Deaths	53	49	102	$\chi^2 = 0\cdot 94150 \quad P = \cdot 8122$
Total	146	161	307	

These figures can be divided into two sub-groups, (1) Natives who had previously received prophylactic inoculation, and (2) those who had not. In 110 cases prophylactic inoculation had been given, and in this group the correlation is suggestive that treatment with the vaccine was actually harmful.

TABLE LXIV.

	Vaccine treatment.			
	+	—	Total.	
Recoveries	29	48	77	$r_t = -·3415 \pm ·0976$
Deaths	20	13	33	$\chi^2 = 4·37980 \quad P = ·2274$
Total	49	61	110	

Of the previously uninoculated Natives there is no evidence that vaccine treatment had any effect in influencing the case mortality from this disease; thus,

TABLE LXV.

	Vaccine treatment.			
	+	—	Total.	
Recoveries	64	64	128	$r_t = ·0335 \pm ·0775$
Deaths	33	36	69	$\chi^2 = 0·08948 \quad P = ·9822$
Total	97	100	197	

Unfortunately no discoverable system in regard to number of doses, size of dose, or period of the disease at which the doses were given was adopted, so that it is impossible to sub-divide these groups further with any advantage. So far as the results of this therapeutic experiment go we can say that there is no evidence that vaccine treatment was of any value, and in the cases in which a prophylactic dose of vaccine had been previously received there is a certain amount of evidence to suggest that actual harm resulted from such treatment.

I do not think, however, that it would be wise to generalise too much from these results, and probably a large amount of more careful work will have to be done before any final opinion on this question can be satisfactorily arrived at. These results are, nevertheless, somewhat surprising in view of the definite statements which have, from time to time, been made in respect to the efficacy of this particular form of treatment. For example, Dr. J. Eyre[19] writes as follows:—" Turning now to the other phase of bacterial therapy, the employment of ' killed cultivations ' or ' vaccines ' in the treatment of pneumonia, a considerable measure of success is obtained." Also in the same article, after having described the method of dosage with pneumococcal vaccine, the author proceeds thus:—"By this means the marked rise in available opsonin that naturally precedes the crisis, and the consequent fall in temperature and disappearance of physical signs, may be considerably anti-dated, and the process of cure correspondingly hastened."

The therapeutic inoculations here discussed were carried out entirely under Sir Almroth Wright's personal supervision, and by his assistants, so that it is unlikely that errors in technique can account for the want of success obtained in these cases. No figures are recorded in support of Eyre's assertions, so that it is impossible to hazard any suggestions as to why the results recorded here correspond so badly with those claimed by some European writers.

SECTION V.

General Conclusions.

The conclusions to be drawn from the foregoing investigation may now be briefly stated.

From Section I.:—

(1) That the attack-rate and death-rate from pneumonia amongst "Tropical Natives" living on the Rand are influenced by the country of origin from which the Natives are recruited.

(2) That the case mortality is apparently independent of the country of origin.

(3) That the highest pneumonia attack-rates are found among the gangs which—judged by the number of "detentions" and "rejects"—have the lowest physique.

(4) That the greatest incidence of pneumonia occurs immediately on the arrival of the Natives on the Rand, and decreases as the period of their residence increases.

(5) That the case mortality so far as can be judged from a six-months record is apparently uninfluenced by the length of residence on the Rand.

(6) That pneumonia is most prevalent during the cold season.

From Section II.:—

(7) That the clinical signs and symptoms of pneumonia among "Tropical Natives" do not differ essentially from those among Europeans.

(8) That the length of the febrile period of the disease is probably somewhat shorter among these Natives than among Europeans in the Northern Hemisphere.

(9) That while the methods of the termination of the pyrexial stage are similar to those observed elsewhere, the percentage of cases which terminate by "crisis" is, among these Natives, lower than the percentages recorded for Europeans in other countries.

(10) That there does not appear to be any essential difference in the clinical characters of the attack, nor in the length of the convalescence, whether the fever terminate by "crisis" or by "lysis."

(11) That "second attacks" of pneumonia (as defined in this memoir) are most common immediately after the termination of the first attack, and become progressively more rare as the period after the first increases.

(12) That the rate of "second attacks" among the population of the previously attacked is higher than the primary attack-rate among the whole population.

(13) That the case mortality of "second attacks" is probably lower than that of "first attacks."

(14) That pneumococcal meningitis as a complication of pneumonia appears to be considerably more common among "Tropical Natives" than among Europeans.

(15) That amongst the Natives it is more common to find two or more lobes involved in the pneumonic process than amongst Europeans.

(16) That the stage of hepatization of the lung, as observed at the post-mortem, bears no constant relation to the length of the illness before death.

(17) That the most common day for death to take place in pneumonia is the fourth day of illness.

Section III.:—

(18) That there is no evidence from the data examined that pneumonia spreads from case to case.

(19) That the irregular grouping which is observed in some of the tables cannot be satisfactorily accounted for on the assumption that this disease is infectious.

Section IV.:—

(20) That prophylactic inoculation with a pneumococcal vaccine, when employed immediately on the arrival of these Natives on the Rand, appears to reduce the incidence of pneumonia for a limited period.

(21) That the protective value is greatest immediately or shortly after inoculation, and progressively diminishes.

(22) That its effect does not appear to last for more than about four months.

(23) That in these records there is no evidence that prophylactic inoculation has any influence on the case mortality, except possibly for a very short time after the injection.

(24) That the experimental treatment of cases with pneumococcal vaccine did not favourably influence the mortality from the disease.

(25) That there is some evidence to suggest that treatment with pneumococcal vaccine of cases which had recently received a prophylactic injection of a similar vaccine was harmful.

The investigation has thrown but little light on the problem of the causes which determine the outbreak of pneumonia among new arrivals on the Reef; further information is necessary before a satisfactory hypothesis can be propounded. I have recently examined the nasal secretions of a small number of "East Coast" Natives on their arrival on the Rand, with a view to determining whether the pneumococcus is present at this time. The results so far, although a sufficient number have not yet been examined to enable rates to be satisfactorily determined, show that it is not uncommon to find a pneumococcus in the naso-pharynx of these Natives within a few hours of their arrival in the W.N.L.A. Compound. The obvious question which now requires an answer is—does the pneumococcus exist in the naso-pharynx of these Natives when in their homes, and if not, at what point on their journey to the mines do they become infected?

It has been pointed out in Section I. that the outbreak of pneumonia synchronises with the arrival of the Natives on the Rand, and a similar phenomenon has been observed elsewhere when a change of residence is made. It is of considerable practical importance to determine whether change of climate is accompanied by the appearance of the pneumococcus in a previously uninfected naso-pharynx, or whether there be no increase in the rate of such infections owing to the change in locality. If this latter event were found to be correct, and the increased incidence of pneumonia be not associated with a greater prevalence of the pneumococcus in the throats of the apparently healthy, then the suggestion that the most important factor in determining the occurrence of the disease is some change in the body of the immigrant—such as a lowered resistance—brought about by the alterations in climate would gain support.

There seems little doubt that pneumonia is not unknown, and possibly not even rare, among " Tropical Natives " in their own country. It is notoriously difficult to get reliable information from uneducated Natives. By cross-examining Natives from different parts of the tropical recruiting area, I have obtained an account of a disease which has the symptoms of pain in the chest, spitting of blood-stained sputum, and shortness of breath. This disease is stated to be frequently fatal within three or four days of its onset. Some witnesses state that death may even take place in the first day of illness. Women and children, as well as men, are reported to suffer from it; this is important, as it shows that the disease is not confined to males who have returned from the mines. It is variously stated to be " common " and " not very common " by Natives from different districts. Mozambique and Nyassaland Natives give the native name as " Parare " (literally, pain under the ribs); known by the Maganja da Costa Natives as " Aparu "; by the Yao's as " Chisorma "; Negroes from Blantyre district call it " Kalaso "; B.N.P. name " Chirombo."

These witnesses have uniformly stated, " the disease is a serious one." Although no doubt other diseases may be included under such names, the description given undoubtedly suggests that cases of pneumonia do occur in these districts.

The Secretary of the W.N.L.A. has kindly given me permission to publish the following interesting letter on this subject from Mr. Hanner.

Witwatersrand Native Labour Association, Limited.
Matolla Agency,
18th November, 1913.

The Secretary,
W.N.L.A., Ltd.,
Johannesburg.

Dear Sir,

Re Pneumonia amongst Tropicals.

In reply to yours of the 14th inst. referring to the above, I beg to inform you that a disease very similar to pneumonia is quite common amongst Nyassa and Zambesi Natives.

In Portuguese Nyassaland it is known as "Ngonjua Chifua," or "Kossamolla," and amongst the Natives of the Zambesi valley I believe it is called "Anacoshola Benca," but as it is some sixteen or seventeen years since last I had occasion to speak "Chikunda" I would not be positive as to whether the latter name is correct.

As far as my experience goes I think that chest diseases are more prevalent amongst the Natives of the Upper Lujela (Quilimane district). It may be of interest to you to know that some 25 years ago, when I first went to the Zambesi, they had a tremendous famine which literally wiped out the population by tens of thousands. I myself have seen people lying dead under the trees all over the country, and also in their villages. It became so bad at Tete that they brought the bodies and threw them just outside the cemetery.

The tribe that suffered the most were the people who lived north of Quilimane and towards "Shirwa," and I think that this accounts for the poor physique of the Natives living in this part of the country.

The people who suffer the least from chest diseases are a tribe called "Azimba," who live in a range of mountains some 20 miles north of the "Kebrabassa rapids," and which range runs practically up to "Undis." These people are of a splendid physique, but very wild. When I was living amongst them the paramount Chief was named "Biwi." It may be that these people are not so susceptible to chest complaints, owing to the high altitude at which they live.

Yours faithfully,
(Signed) R. M. HANNER,
Div. Recruiter.

Clearly this hearsay evidence requires confirmation by medical and bacteriological investigation in the districts themselves, not only in regard to the existence of the disease in these parts, but also as to its relative prevalence.

It has already been mentioned that some of the apparent anomalies in regard to the etiology and manifestations of pneumonia may be explicable on the hypothesis that there are several strains or varieties of the pneumococcus with different properties. If this suggestion receives support by subsequent investigation, then the problem of the cause of the prevalence of pneumonia on the Rand will also require investigation from this point of view. It will be necessary to determine whether the type of organism which produces the disease in tropical districts—if the disease does occur there—is the same as that which is common on the Rand, and if not, is the alteration due to a change in type of pneumococcus developed owing to changes in the host, or again, is a freshly acquired organism the cause of the outbreak? If the pneumococcus be found to be much less prevalent in the low-veld than among Natives arriving on the Rand, then it is important to discover at what point in their journey the greater prevalence is acquired. Arrangements are now being made by the Institute to undertake at an early date an investigation on these lines, to examine the Natives at the various compounds on the route of their journey to the Rand, to inspect them in their kraals, and also if possible to study some cases of pneumonia in the native districts.

Since the foregoing sections have been in the press my attention has been drawn to a paper by Drs. Dochez and Gilespie[27] on the differentiation of strains of pneumococci. In view of this work, and of its confirmation by Dr. Lister in this Institute from a somewhat different standpoint, it will be important to discover whether " second attacks " of pneumonia are due to a different strain of organism. If, as seems possible, this may be so, it will be necessary to determine whether the " second attack " is a new infection, a latent infection becoming virulent owing to the effects of the previous illness, or a variation of the original strain arising by selection. Another problem of importance is to determine whether the pneumococcus found in normal throats is the source of infection to its bearer who develops pneumonia, and if so, whether any discoverable change in its type precedes the attack.

The present investigation will, I hope, serve a useful purpose by having established certain facts in the etiology of this disease among " Tropical Natives," and by having directed attention to some of the lacunae in our knowledge. Continued scientific investigation is required before any further advance in the production of an artificial immunity against attack, or any solution to the problem of increasing the resistance of the attacked, can be expected. The wholesale application of empiric remedies cannot but have a reactionary effect, and the tendency in some quarters on the Reef to consider the problem of pneumonia as settled will only result in retarding progress.

In conclusion I would like to take this opportunity of thanking Mr. B. G. Lloyd, General Manager of the Witwatersrand Native Labour Association, for his courtesy in giving me free access to the returns of the Association, and Dr. G. A. Turner, Medical Officer to the Association, for a similar favour in connection with the hospital reports and records, without which this investigation could never have been undertaken. My thanks are also due to Dr. J. McD. Troup for much asisstance, particularly in regard to some of the mathematical problems in Section III.; and to Dr. W. Watkins-Pitchford, the Director of the Institute, for constant help and advice in the numerous discussions which arose in the course of the investigation, and for his kindness in reading through the manuscript. I would also take this opportunity to express my appreciation of the painstaking and conscientious manner in which Mr. P. Hardwick has carried out the laborious duty of compiling and checking the returns of the inoculation experiments.

Finally, I wish to state, that although I have sometimes had occasion to adversely criticise statements made in the leading text-books of Medicine, yet this has not been done in a captious spirit; but because such criticisms were essential in order to show that the statements had not been disregarded. No thoughtful practitioner can be insensible of the debt the profession owes to the authors of these books, nor can he fail to realise the difficulty of maintaining every portion of such works at a uniformly high level.

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APPENDIX.

A Pneumococcal Vaccine Inoculation Experiment on Natives in the employ of the Central Mining and Rand Mines Groups.

Since the manuscript of the foregoing sections has been in the press the following tables, summarising the results obtained from an experiment in anti-pneumonia vaccination, have been issued. This experiment has been carried out on the mines under the control of the Central Mining and Rand Mines Groups, and my thanks are due to the managements for permission to publish the returns.

In consultation with the Medical Officers of these mines it was decided to inoculate three-quarters of all " Tropical," " East Coast," and Basuto Natives on the properties, the remaining quarter to be left as controls to the larger group. Before being inoculated the Natives were grouped according to the length of their previous service on the mine, and every fourth " boy " excluded from the inoculation, and classed as a control. Only one inoculation was given, the dose being " 1000 millions." The results of this experiment, for the first four months, are reproduced in Tables A., B., C., and D.

It is unnecessary to lay stress on the fact that in an investigation such as the present, where many managements are concerned in carrying out a somewhat complicated procedure, the experimental errors will necessarily tend to be larger than when the whole experiment is conducted by one set of persons. It would appear, however, that on many of the mines, and of these some are amongst the largest employers of native labour in the groups, the greatest care was taken to carry out minutely the instructions issued, while it is equally certain that on others much less trouble was taken.

On one large group of mines the Medical Officers declined to be informed as to whether a patient was or was not an inoculated " boy " until he left hospital, in order that no unconscious bias should affect their diagnosis. This is an example of a detail in method which might with advantage be followed by all medical men who desire to obtain a true estimate of the facts of such experiments. No very extensive knowledge of human nature is required to establish that the presence of a bias, even amongst those who are most desirous of remaining strictly impartial, is inevitable where an observer has any opinion as to the efficacy or otherwise of any procedure the effect of which he is investigating. Further, no one who has taken an interest in a subject and heard it discussed, can but have formed some mental estimate of its value, however tentatively such an estimate may be held. This digression has been made, because the effect of an unconscious bias does not appear to be sufficiently recognised, and, further, because some of the reports received would have had a considerably increased value had the method adopted by these Medical Officers been followed by their authors.

Nevertheless, so far as can be ascertained there does not appear to be any special source of error in regard to this experiment which would apply to the inoculated more than to the "controls," or *vice versa*.

Turning to an examination of the data given in Tables A, B, C, D, appearing at end of text, the effect of inoculation may be studied by the same methods as used in Section IV.; but here the calculation of the correlation from the "four-fold table" will not lead to any useful purpose. It will be sufficient to calculate the "goodness of fit" of observation to theory on the basis of independence. The following four tables are constructed from the group "all Natives" for each of the four months, and deal with the number attacked among the "inoculated" and the "controls."

TABLE F. (First Month).

	Inoculation.			
	+	—	Total.	
Not attacked ...	15618	5183	20801	$\chi^2 = 0{\cdot}047876$
Attacked	83	29	112	$P = {\cdot}9905$
Total	15701	5212	20913	

TABLE G. (Second Month).

	Inoculation.			
	+	—	Total.	
Not attacked ...	14883	4900	19783	$\chi^2 = 0{\cdot}149388$
Attacked	55	16	71	$P = {\cdot}9703$
Total	14938	4916	19854	

TABLE H. (Third Month).

	Inoculation.			
	+	—	Total.	
Not attacked ...	13883	4577	18460	$\chi^2 = 4{\cdot}346127$
Attacked	39	22	61	$P = {\cdot}2304$
Total	13922	4599	18521	

TABLE I. (FOURTH MONTH).

	Inoculation.			
	+	—	Total.	
Not attacked	12980	4314	17294	$\chi^2 = 3{\cdot}080320$
Attacked	38	6	44	$P = {\cdot}3812$
Total	13018	4320	17338	

In the first month the attack-rates were actually slightly in favour of the "controls," and in the second in favour of the "inoculated," but clearly neither value has any significance. In the third month the balance in favour of the inoculated is more marked, and the chance against a divergence as great as that observed having arisen from errors of sampling is one to four. In the fourth month the lower attack-rate is again found amongst the uninoculated.

Averaging the population for the four months, the following table is obtained:—

TABLE J.

	Inoculation.			
	+	—	Total.	
Not attacked	14180	4689	18869	$\chi^2 = 0{\cdot}018802$
Attacked	215	73	288	$P = {\cdot}9963$
Total	14395	4762	19157	

Thus from these returns, employing the group "All Natives" as the measure of efficacy, no support is obtained for the hypothesis that the pneumococcal vaccination had any effect in lowering the attack-rate from pneumonia. Can we therefore *legitimately assert* that these figures prove that the prophylactic use of this vaccine is without value? I think not. The attack-rates from this disease were comparatively speaking low throughout the period, and, therefore, a real protection might be masked by the errors of sampling. The probability of such an occurrence can be investigated in the following way. We may assume that a certain percentage reduction in the attack-rate should follow inoculation, and then by regarding the attack-rate of either group as representing the normal for that group we can calculate the rate to be expected in the other group, and from it the most probable number of Natives who would, on this hypothesis, have contracted the disease. The distribution thus found is then regarded as the "most probable," and the deviation from it as being due to errors of sampling. In the following tables I have regarded the observed rate of each group in turn as the "normal"; the true normal rate would probably lie between these two values, and therefore the value of P should lie between the values as given in the tables.

TABLE K. (First Month).

Basis of 30 per cent. reduction due to Inoculation.

Normal rate 5·6 per thousand.

∴ rate among Inoculated 3·9 per thousand.

Distribution expected.

+	—
15640	5183
81	29

$\chi^2 = 7{\cdot}9652$

$P = {\cdot}0469$

Normal rate 5·3 per thousand

∴ rate among "Controls" 7·6 per thousand.

Distribution expected.

+	—
15618	5172
83	40

$\chi^2 = 3{\cdot}0484$

$P = {\cdot}3853$

TABLE L. (Second Month).

Basis of 20 per cent. reduction due to Inoculation.

Normal rate 3·3 per thousand.

∴ rate among Inoculated 2·6 per thousand.

Distribution expected.

+	—
14899	4900
39	16

$\chi^2 = 7{\cdot}546618$

$P = {\cdot}0577$

Normal rate 3·7 per thousand.

∴ rate among "Controls" 4·6 per thousand.

Distribution expected.

+	—
14883	4893
55	23

$\chi^2 = 2{\cdot}140451$

$P = {\cdot}5470$

TABLE M. (Third Month).

Basis of 10 per cent. reduction due to Inoculation.

Normal rate 4·8 per thousand.

∴ rate among Inoculated 4·3 per thousand.

Distribution expected.

+	—
13862	4577
62	22

$\chi^2 = 7{\cdot}381818$

$P = {\cdot}0620$

Normal rate 2·8 per thousand.

∴ rate among "Controls" 3·1 per thousand.

Distribution expected.

+	—
13883	4585
39	14

$\chi^2 = 4{\cdot}585386$

$P = {\cdot}2090$

TABLE N. (TOTAL FOR FOUR MONTHS).

Basis of 15 per cent. reduction due to Inoculation.

Normal rate 15·3 per thousand.
∴ rate among Inoculated 13·0 per thousand.

Distribution expected.

+	—
14198	4689
187	73

$\chi^2 = 4·247731$
$P = ·2393$

Normal rate 14·9 per thousand.
∴ rate among "Controls" 17·5 per thousand.

Distribution expected.

+	—
14180	4679
215	83

$\chi^2 = 1·226191$
$P = ·7495$

This method of considering the problem will only give us a rough guide to the improbability that the observed results have arisen from a population which, if large enough to render the errors of sampling negligible, would have shown the percentage reduction assumed in the tables. The right hand table of each pair is based on the more reasonable assumption, because the rates calculated from the larger group will be less influenced by errors of sampling; clearly, however, neither table is more than a rough approximation, as the rate in the group called "normal" is considered as being free from errors of sampling. The results, nevertheless, seem to me to indicate that we cannot safely conclude that these returns *prove* pneumococcal vaccination, even in this experiment, to be valueless; for variations such as those observed might have arisen from errors of sampling, even if the percentage reductions such as those assumed were to have been expected. It will be necessary, therefore, to wait for larger samples—when such small total rates occur amongst the attacked—before dogmatising on this matter, and I do not think this experiment should be accepted as definitely negativing the results found from the "8000 experiment." All we can assert is, that *this experiment*, carried out on the mines among a group of Natives heterogeneous as to race and length of service, do not confirm the results deduced from the "8000 experiment." We can, however, from the tables supplied, get rid of these sources of heterogenity, and the following tables deal with the results for the four months for each territorial group. Further sub-division has not been undertaken owing to the small number of cases which occur in sub-groups, except in the case of the group "All Natives," where the values of χ^2 and P are shown for the sub-groups according to period of service.

TABLE O.

Basutos (four months).

	Inoculation.			
	+	—	Total.	
Not attacked ...	923	305	1228	$\chi^2=0{\cdot}000000$
Attacked	15	5	20	$P=1{\cdot}$
Total	938	310	1248	

TABLE P.

"East Coast" (four months).

	Inoculation.			
	+	—	Total.	
Not attacked ...	9612	3213	12825	$\chi^2=0{\cdot}348257$
Attacked	100	38	138	$P={\cdot}9308$
Total	9712	3251	12963	

TABLE Q.

"Tropicals" (four months).

	Inoculation.			
	+	—	Total.	
Not attacked ...	3645	1172	4817	$\chi^2=0{\cdot}174364$
Attacked	100	29	129	$P={\cdot}9653$
Total	3745	1201	4946	

The tables of the sub-groups of the group "All Natives," classed according to period of service on the mines before inoculation, are as under:—

TABLE R.

Under three months' service.

	Inoculation.			
	+	—	Total.	
Not attacked ...	3901	1220	5121	$\chi^2=0{\cdot}046428$
Attacked	93	28	121	$P={\cdot}9908$
Total	3994	1248	5242	

TABLE S.

Under six months' service.

	Inoculation.			
	+	—	Total.	
Not attacked	4138	1357	5495	$\chi^2 = 1{\cdot}479008$
Attacked	63	28	91	$P = {\cdot}6916$
Total	4201	1385	5586	

TABLE T.

Under nine months' service.

	Inoculation.			
	+	—	Total.	
Not attacked	2683	866	3549	$\chi^2 = 0{\cdot}428097$
Attacked	37	11	48	$P = {\cdot}9149$
Total	2720	877	3597	

TABLE U.

Over nine months' service.

	Inoculation.			
	+	—	Total.	
Not attacked	3457	1246	4703	$\chi^2 = 0{\cdot}191569$
Attacked	22	6	28	$P = {\cdot}9619$
Total	3479	1252	4731	

It is evident, therefore, that the absence of any indication of the efficacy of vaccination as a prophylactic measure is not due to the masking of the real effect due to the heterogeneity of the group "All Natives." In none of the sub-groups examined in Tables O. to U. is there any evidence that the inoculated Natives had an appreciably lower attack-rate than the "controls."

Finally, it may be remarked that these tables illustrate once more the fall in the attack-rate with increase in the period of residence on the Reef. The total attack-rate for the four months for Natives, under three months is 23 per thousand, under six months 16 per thousand, under nine months 13 per thousand, and over nine months 6 per thousand.

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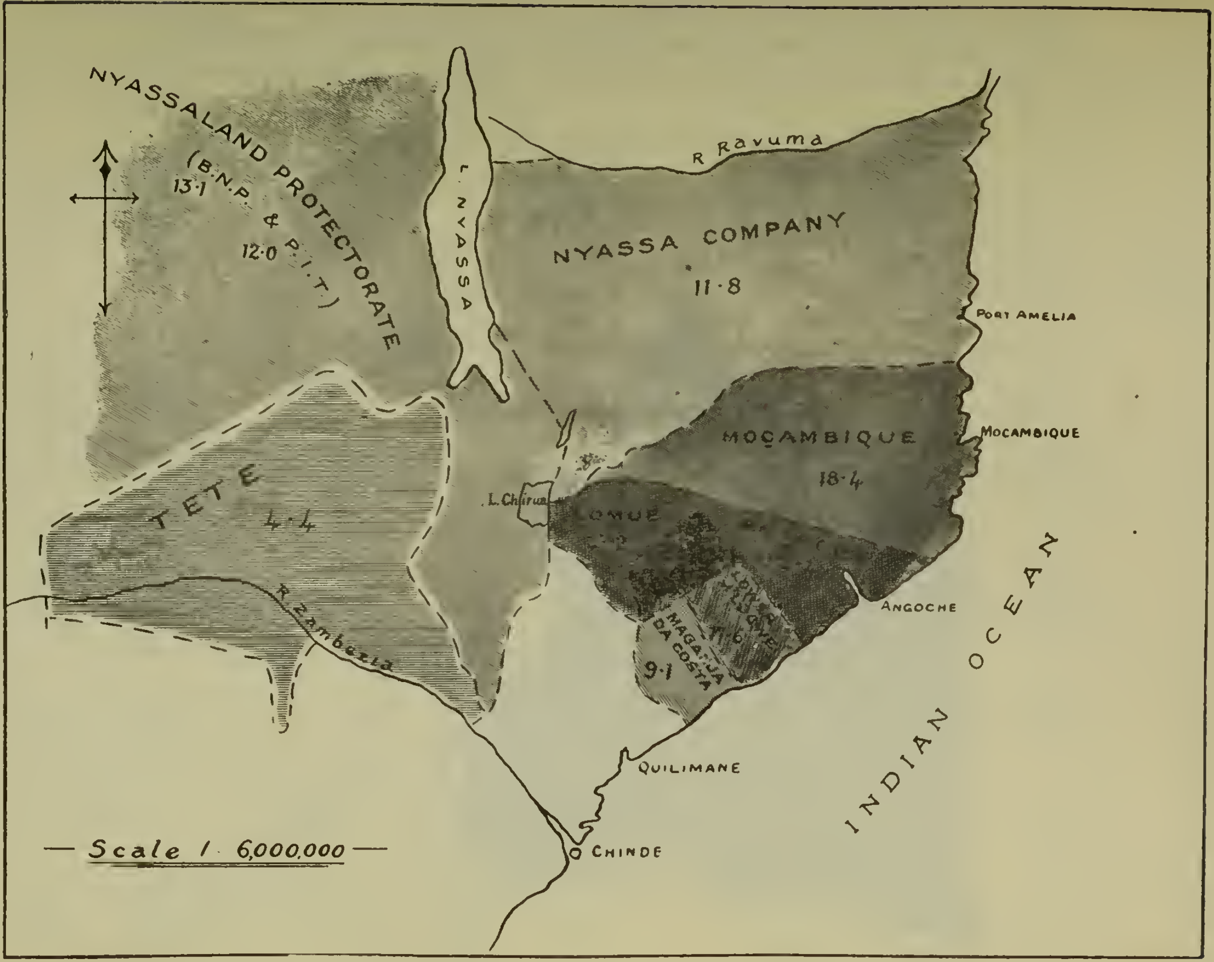
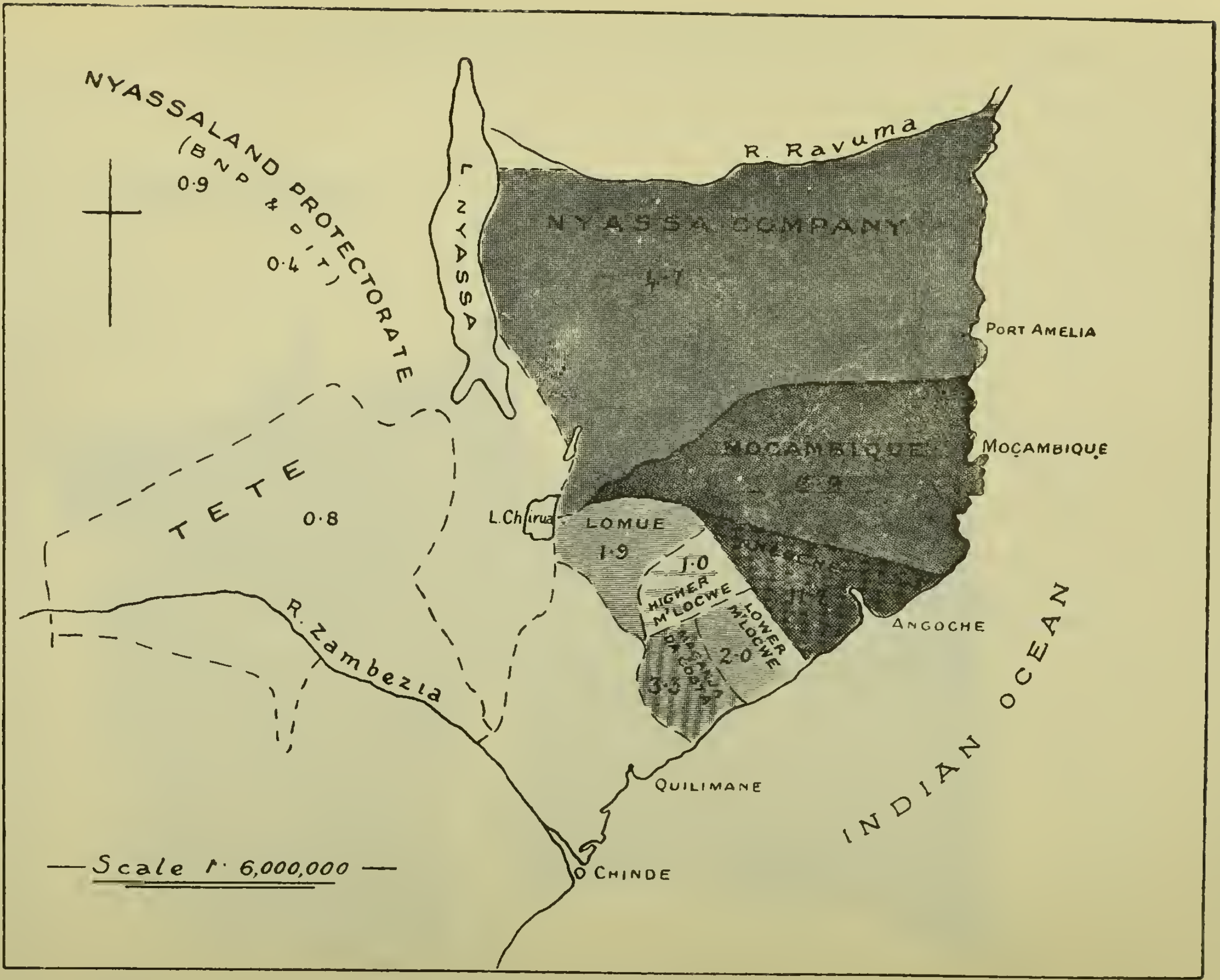
SKETCH MAP I.



Showing recruiting areas north of Lat. 22° S. of W.N.L.A., Ltd. Shaded to indicate comparative death-rates on the Rand from Pneumonia.

SKETCH MAP II.



Showing recruiting areas north of Lat. 22° S. of W.N.L.A., Ltd. Shaded to indicate comparative death-rates on the Rand from Cerebro-spinal Meningitis.

A

CENTRAL MINING—RAND MINES GROUP.

PNEUMOCOCCIC VACCINE INOCULATION EXPERIMENT ON NATIVES.

AMENDED RETURN FOR FIRST MONTH AFTER INOCULATION.

Class of Native.	INOCULATED NATIVES.										CONTROL NATIVES.									
	No. of Natives originally inoculated.	Average No. of inoculated Natives in Co's service during 1st month succeeding inoc'n.	PNEUMONIA.				OTHER RESPIRATORY DISEASES.				No. of Natives originally set aside as controls.	Average No. of Control Natives in Co.'s service during 1st month.	PNEUMONIA.				OTHER RESPIRATORY DISEASES.			
			ADMISSIONS TO HOSPITAL.		DEATHS		ADMISSIONS TO HOSPITAL.		DEATHS.				ADMISSIONS TO HOSPITAL.		DEATHS.		ADMISSIONS TO HOSPITAL.		DEATHS.	
			No.	Rate per 1,000 per month	No.	Rate per 1,000 per month.	No.	Rate per 1 000 per month	No.	Rate per 1,000 per month			No.	Rate per 1,000 per month	No.	Rate per 1,000 per month	No.	Rate per 1,000 per month	No.	Rate p r 1,000 per month
EAST COAST.—																				
Under 3 months' service	2,279	2,105	11	5·2	3	1·4	10	4·8			700	639	3	4·7			4	6·3		
From 3—6 ,, ,,	2,869	2,736	6	2·2			7	2·6			988	933	3	3 2	1	1·1	8	8·6		
,, 6—9 ,, ,,	2,199	2,088	6	2·9	1	·5	2	1·0			732	693	6	8·7			1	1·4		
Over 9 ,, ,,	3,848	3,413	7	2·1	2	·6	2	·6			1,369	1,220					1	·8		
Totals and Averages	11,195	10,342	30	2·9	6	·6	21	2·0			3,789	3,485	12	3 4	1	·3	14	4·0		
TROPICALS.—																				
Under 3 months' service	1,516	1,454	22	15·1	4	2 8	7	4·8			471	456	6	13·2	2	4·4	2	4·4		
From 3—6 ,, ,,	1,535	1,471	18	12·2	4	2·7	7	4·8			494	485	6	12 4			1	2·1		
,, 6—9 ,, ,,	804	768	6	7·8	1	1·3	4	5·2			261	248	1	4·0			4	16·1	1	4·0
Over 9 ,, ,,	559	402					1	2 5			169	130								
Totals and averages	4,414	4,095	46	11·2	9	2·2	19	4 6			1,395	1,319	13	9·9	2	1·5	7	5·3	1	·8
BASUTOS.—																				
Under 3 months' service	915	786	7	8·9			1	1·3			285	248	4	16·1			1	4·0		
From 3—6 ,, ,,	341	268									118	91								
,, 6—9 ,, ,,	90	48									27	14								
Over 9 ,, ,,	190	162									63	55								
Totals and Averages	1,536	1,264	7	5·5			1	·8			493	408	4	9·8			1	2·5		
ALL NATIVES—																				
Under 3 months' service	4,710	4,345	40	9·2	7	1 6	18	4·1			1,456	1,343	13	9 7	2	1·5	7	5·2		
From 3—6 ,, ,,	4,745	4,475	24	5·4	4	·9	14	3·1			1,600	1,509	9	5·6	1	·6	9	6·0		
,, 6—9 ,, ,,	3,093	2,904	12	4 1	2	·7	6	2·1			1,020	955	7	7·3			5	5·2	1	1·0
Over 9 ,, ,,	4,597	3,977	7	1 8	2	·5	3	·8			1,601	1,405					1	·7		
Grand Totals and Averages	17,145	15,701	83	5·3	15	1·0	41	2·6			5,677	5,212	29	5·6	3	·6	22	4·2	1	·2

B

CENTRAL MINING—RAND MINES GROUP.

PNEUMOCOCCIC VACCINE INOCULATION EXPERIMENT ON NATIVES.

RETURN FOR SECOND MONTH AFTER INOCULATION.

Class of Native.	INOCULATED NATIVES.										CONTROL NATIVES.									
	No. of Natives originally inoculated.	Average No. of inoculated Natives in Co's service during 2nd month succeeding inoc'n.	PNEUMONIA.				OTHER RESPIRATORY DISEASES.				No. of Natives originally set aside as controls.	Average No. of Control Natives in Co.'s service during 2nd month.	PNEUMONIA.				OTHER RESPIRATORY DISEASES.			
			ADMISSIONS TO HOSPITAL.		DEATHS		ADMISSIONS TO HOSPITAL.		DEATHS.				ADMISSIONS TO HOSPITAL.		DEATHS.		ADMISSIONS TO HOSPITAL.		DEATHS.	
			No.	Rate per 1,000 per month	No.	Rate per 1,000 per month.	No.	Rate per 1,000 per month	No.	Rate per 1,000 per month			No.	Rate per 1,000 per month	No.	Rate per 1,000 per month	No.	Rate per 1,000 per month	No.	Rate per 1,000 per month
EAST COAST.—																				
Under 3 months' service	2,279	2,073	9	4·3			9	4·3	1	·5	700	636	1	1·6			2	3·1		
From 3—6 ,, ,,	2,869	2,740	8	2·9	2	·7	7	2·6			988	904	5	5·5	1	1·1	4	4·4	1	1·1
,, 6—9 ,, ,,	2,199	2,111	4	1·9	1	·5	4	1·9	1	·5	732	678								
Over 9 ,, ,,	3,848	3,157	7	2·2			7	2·2	2	·6	1,369	1,137	2	1·8	1	·9	2	1·8		
Totals and Averages	11,195	10,081	28	2·8	3	·3	27	2·7	4	·4	3,789	3,355	8	2·4	2	·6	8	2·4	1	·3
TROPICALS.—																				
Under 3 months' service	1,516	1,377	7	5·1	1	·7	5	3·6			471	438	2	4·6						
From 3—6 ,, ,,	1,535	1,349	7	5·2			4	3·0			494	438	3	6·8	1	2·3				
,, 6—9 ,, ,,	804	745	6	8·0	2	2·7					261	237	1	4·2						
Over 9 ,, ,,	559	306	1	3·3			1	3·3			169	102								
Totals and averages	4,414	3,777	21	5·6	3	·8	10	2·6			1,395	1,215	6	4·9	1	·8				
BASUTOS.—																				
Under 3 months' service	915	709	4	5·6	2	2·8	1	1·4			285	220	1	4·5	1	4·5	1	4·5		
From 3—6 ,, ,,	341	198									118	71								
,, 6—9 ,, ,,	90	39									27	11								
Over 9 ,, ,,	190	134	2	14·9							63	44								
Totals and Averages	1,536	1,080	6	5·6	2	1·9	1	·9			493	346	1	2·9	1	2·9	1	2·9		
ALL NATIVES—																				
Under 3 months' service	4,710	4,159	20	4.8	3	·7	15	3·6	1	·2	1,456	1,294	5	3·9	1	·8	3	2·3		
From 3—6 ,, ,,	4,745	4,287	15	3·5	2	·5	11	2·6			1,600	1,413	8	5·7	2	1·4	4	2·8	1	·7
,, 6—9 ,, ,,	3,093	2,895	10	3·4	3	1·0	4	1·4	1	·3	1,020	926	1	1·1						
Over 9 ,, ,,	4,597	3,597	10	2·8			8	2·2	2	·6	1,601	1,283	2	1·6	1	·8	2	1·6		
GRAND TOTALS AND AVERAGES	17,145	14,938	55	3·7	8	·5	38	2·5	4	·3	5,677	4,916	16	3·3	4	·8	9	1·8	1	·2

C

CENTRAL MINING—RAND MINES GROUP.

PNEUMOCOCCIC VACCINE INOCULATION EXPERIMENT ON NATIVES.

RETURN FOR THIRD MONTH AFTER INOCULATION

Class of Native.	INOCULATED NATIVES.										CONTROL NATIVES.									
	No. of Natives originally inoculated.	Average No. of inoculated Natives in Co's service during 3rd month succeeding inoc'n.	PNEUMONIA.				OTHER RESPIRATORY DISEASES.				No. of Natives originally set aside as controls.	Average No. of Control Natives in Co.'s service during 3rd month.	PNEUMONIA.				OTHER RESPIRATORY DISEASES.			
			ADMISSIONS TO HOSPITAL.		DEATHS		ADMISSIONS TO HOSPITAL.		DEATHS.				ADMISSIONS TO HOSPITAL.		DEATHS.		ADMISSIONS TO HOSPITAL.		DEATHS.	
			No.	Rate per 1,000 per month	No.	Rate per 1,000 per month.	No.	Rate per 1.000 per month	No.	Rate per 1,000 per month			No.	Rate per 1,000 per month	No.	Rate per 1,000 per month	No.	Rate per 1,000 per month	No.	Rate per 1.000 per month
EAST COAST.—																				
Under 3 months' service	2,279	1,976	8	4·0	1	0·5	5	2·5	1	0·5	700	611	5	8·2			2	3·3		
From 3—6 ,, ,,	2,869	2,660	7	2·6	2	0·8	7	2·6	1	0·4	988	871	5	5·7			2	2·3		
,, 6—9 ,, ,,	2,199	2,014	4	2·0	1	0.5	6	3.0	1	0·5	732	645	1	1·6	1	1·6	1	1·6		
Over 9 ,, ,,	3,848	2,848	4	1·4	1	0·4	3	1·1	1	0·4	1,369	1,045	3	2·9						
Totals and Averages	11,195	9,498	23	2·4	5	0·5	21	2·2	4	0·4	3,789	3,172	14	4·4	1	0·3	5	1·6		
TROPICALS.—																				
Under 3 months' service	1,516	1,345	7	5·2	1	0·7	1	0·7			471	432	5	11·6						
From 3—6 ,, ,,	1,535	1,306	4	3·1			2	1·5			494	422	3	7·1	1	2·4	1	2·4		
,, 6—9 ,, ,,	804	708	4	5·6	1	1·4					261	220								
Over 9 ,, ,,	559	257					1	3·9			169	84					1	11.9		
Totals and averages	4,414	3,616	15	4·1	2	0·6	4	1·1			1,395	1,158	8	6·9	1	0·9	2	1.7		
BASUTOS.—																				
Under 3 months' service	915	566	1	1·8			1	1·8			285	177								
From 3—6 ,, ,,	341	130									118	46								
,, 6—9 ,, ,,	90	21									27	10								
Over 9 ,, ,,	190	91									63	36								
Totals and Averages	1,536	808	1	1·2			1	1·2			493	269								
ALL NATIVES—																				
Under 3 months' service	4,710	3,887	16	4.1	2	0·5	7	1·8	1	0·3	1,456	1,220	10	8·2			2	1·6		
From 3—6 ,, ,,	4,745	4,096	11	2·7	2	0·5	9	2·2	1	0·2	1,600	1,339	8	6·0	1	0·7	3	2·2		
,, 6—9 ,, ,,	3,093	2,743	8	2·9	2	0·7	6	2·2	1	0·4	1,020	875	1	1·1	1	1·1	1	1·1		
Over 9 ,, ,,	4,597	3,196	4	1·3	1	0.3	4	1·3	1	0·3	1,601	1,165	3	2·6			1	0·9		
GRAND TOTALS AND AVERAGES	17,145	13,922	39	2·8	7	0·5	26	1·9	4	0·3	5,677	4,599	22	4·8	2	0·4	7	1·5		

D

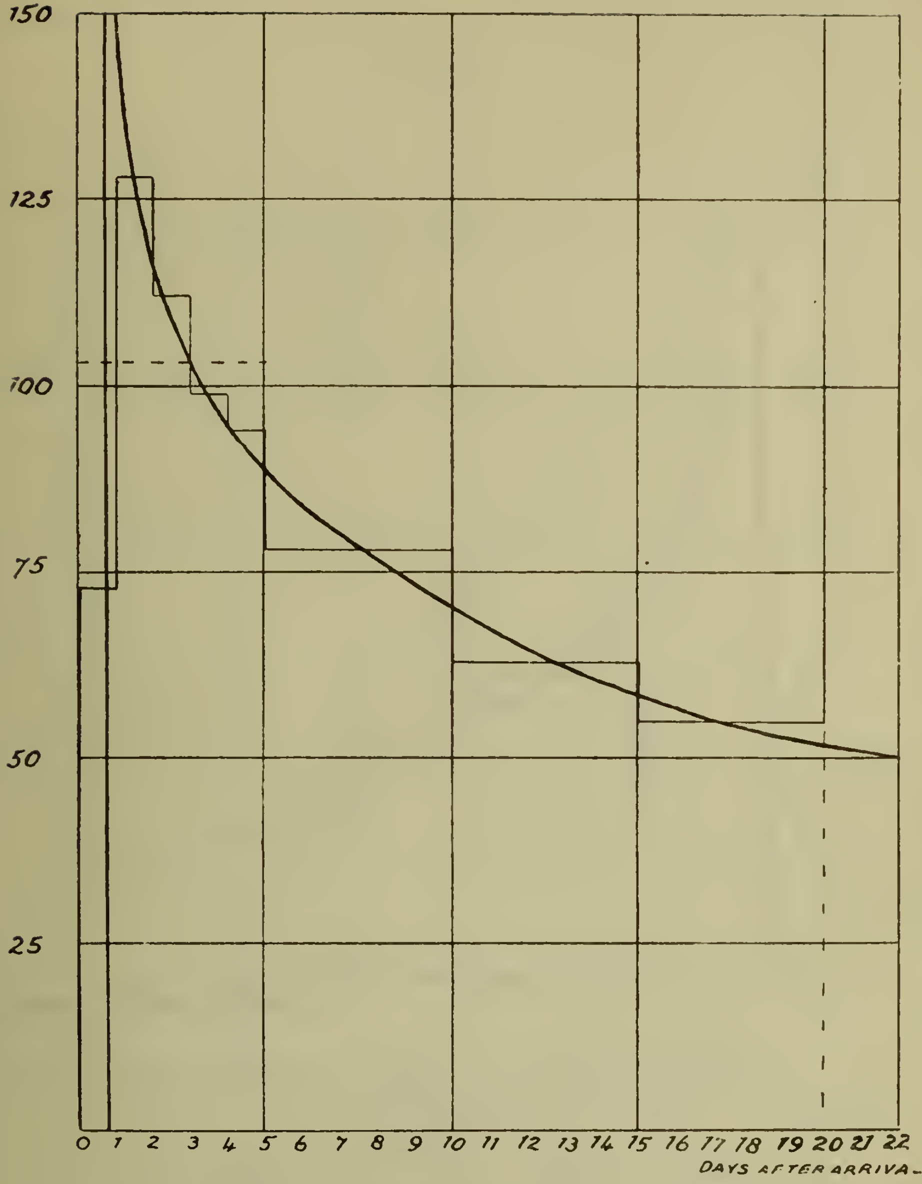
CENTRAL MINING—RAND MINES GROUP.

PNEUMOCOCCIC VACCINE INOCULATION EXPERIMENT ON NATIVES.

RETURN FOR FOURTH MONTH AFTER INOCULATION

Class of Native.	INOCULATED NATIVES.										CONTROL NATIVES.									
	No. of Natives originally inoculated.	Average No. of inoculated Natives in Co's service during 4th month succeeding inoc'n.	PNEUMONIA.				OTHER RESPIRATORY DISEASES.				No. of Natives originally set aside as controls.	Average No. of Control Natives in Co.'s service during 4th month.	PNEUMONIA.				OTHER RESPIRATORY DISEASES.			
			Admissions to Hospital.		Deaths		Admissions to Hospital.		Deaths.				Admissions to Hospital.		Deaths.		Admissions to Hospital.		Deaths.	
			No.	Rate per 1,000 per month	No.	Rate per 1,000 per month.	No.	Rate per 1 000 per month	No.	Rate per 1,000 per month			No.	Rate per 1,000 per month	No.	Rate per 1,000 per month	No.	Rate per 1,000 per month	No.	Rate per 1,000 per month
EAST COAST.—																				
Under 3 months' service	2,279	1,876	10	5·3	1	0·5	3	1·6			700	582					1	1·7		
From 3—6 ,, ,,	2,869	2,572	5	1·9	1	0·4	9	3·5			988	848	2	2·4			1	1·2	1	1·2
,, 6—9 ,, ,,	2,199	1,726	3	1·7	1	0.6	7	4.1			732	540	1	1·9	1	1·9				
Over 9 ,, ,,	3,848	2,755	1	0·4	1	0·4	3	1·1	1	0·4	1,369	1,021	1	1·0			3	2·9		
Totals and Averages	11,195	8,929	19	2·1	4	0·4	22	2·5	1	0·1	3,789	2,991	4	1·3	1	0·3	5	1·7	1	0·3
TROPICALS.—																				
Under 3 months' service	1,516	1,318	7	5·3			7	5·3			471	421					1	2·4		
From 3—6 ,, ,,	1,535	1,272	7	5·5	1	0·8	5	3·9			494	400	1	2·5			1	2·5		
,, 6—9 ,, ,,	804	566	4	7·1	2	3·5	2	3·5			261	191	1	5·2						
Over 9 ,, ,,	559	334									169	102								
Totals and averages	4,414	3,490	18	5·2	3	0·9	14	4·0			1,395	1,114	2	1·8			2	1.8		
BASUTOS.—																				
Under 3 months' service	915	391									285	131								
From 3—6 ,, ,,	341	102	1	9·8							118	31								
,, 6—9 ,, ,,	90	48									27	19								
Over 9 ,, ,,	190	58									63	34					1	29·4		
Totals and Averages	1,536	599	1	1·7							493	215					1	4·7		
ALL NATIVES—																				
Under 3 months' service	4,710	3,585	17	4.7	1	0·3	10	2·8			1,456	1,134					2	1·8		
From 3—6 ,, ,,	4,745	3,946	13	3·3	2	0·5	14	3·5			1,600	1,279	3	2·3			2	1·6	1	0·8
,, 6—9 ,, ,,	3,093	2,340	7	3·0	3	1·3	9	3·8			1,020	750	2	2·7	1	1·3				
Over 9 ,, ,,	4,597	3,147	1	0·3	1	0.3	3	1·0	1	0·3	1,601	1,157	1	0·9			4	3·5		
Grand Totals and Averages	17,145	13,018	38	2·9	7	0·5	36	2·8	1	0·1	5,677	4,320	6	1·4	1	0·2	8	1·9	1	0·2

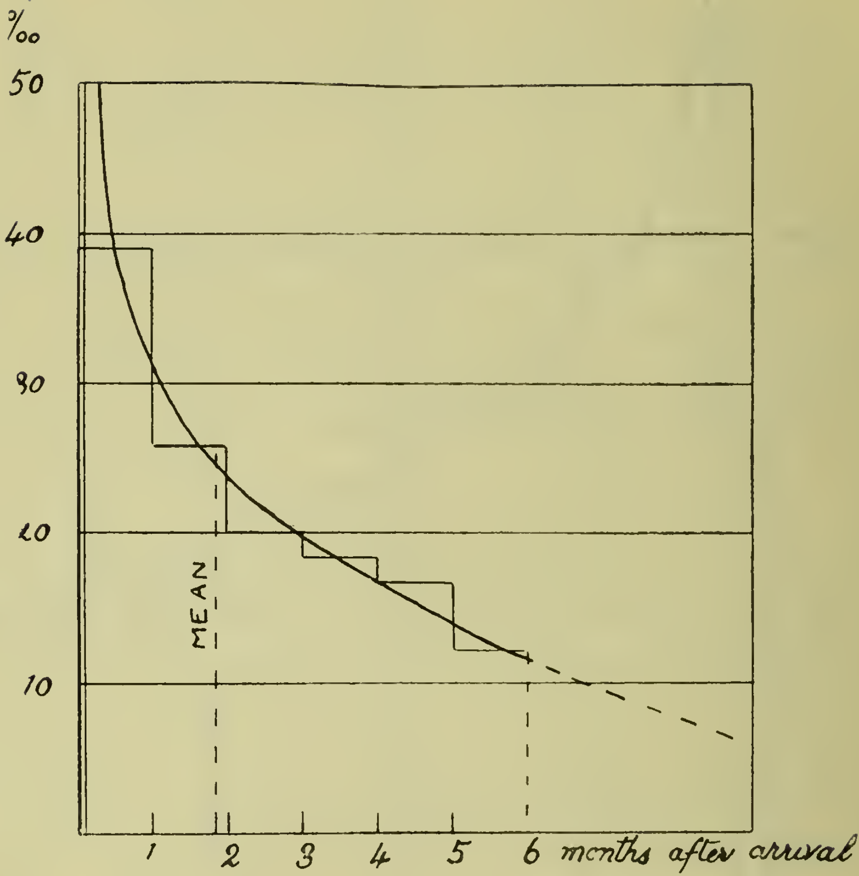
CHART I.



Pneumonia attacks. W.N.L.A. Compound. Tropical Natives. According to number of days after arrival in compound from homes. Population constants for first 21 days.

$$y = 66{\cdot}74\left(\frac{x}{4{\cdot}9276} - 1\right)^{-.2513}\left(1 - \frac{x}{26{\cdot}0598}\right)^{1{\cdot}3290} + 50$$

CHART II.

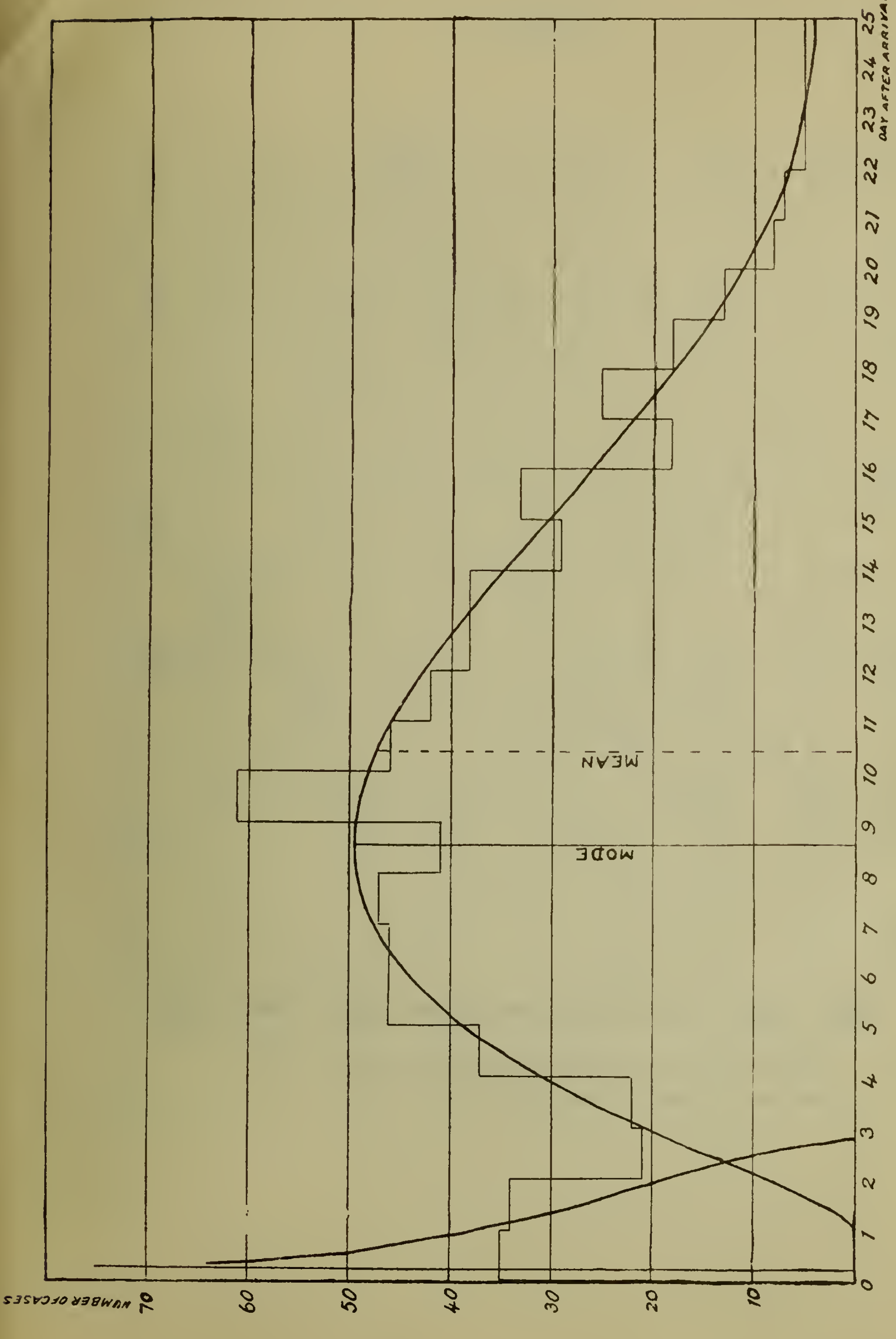


Pneumonia attack-rate per 1,000. For months after arrival on Rand. "Tropical" Natives. [8,123.]

$$y=213{\cdot}3\left(\frac{x}{3{\cdot}8204}-1\right)^{-{\cdot}3012}\left(1-\frac{x}{9{\cdot}9822}\right)^{{\cdot}7870}+10$$

CHART III.

Cerebro-spinal Meningitis. Distribution of cases according to day of onset after arrival in W.N.L.A. Compound.

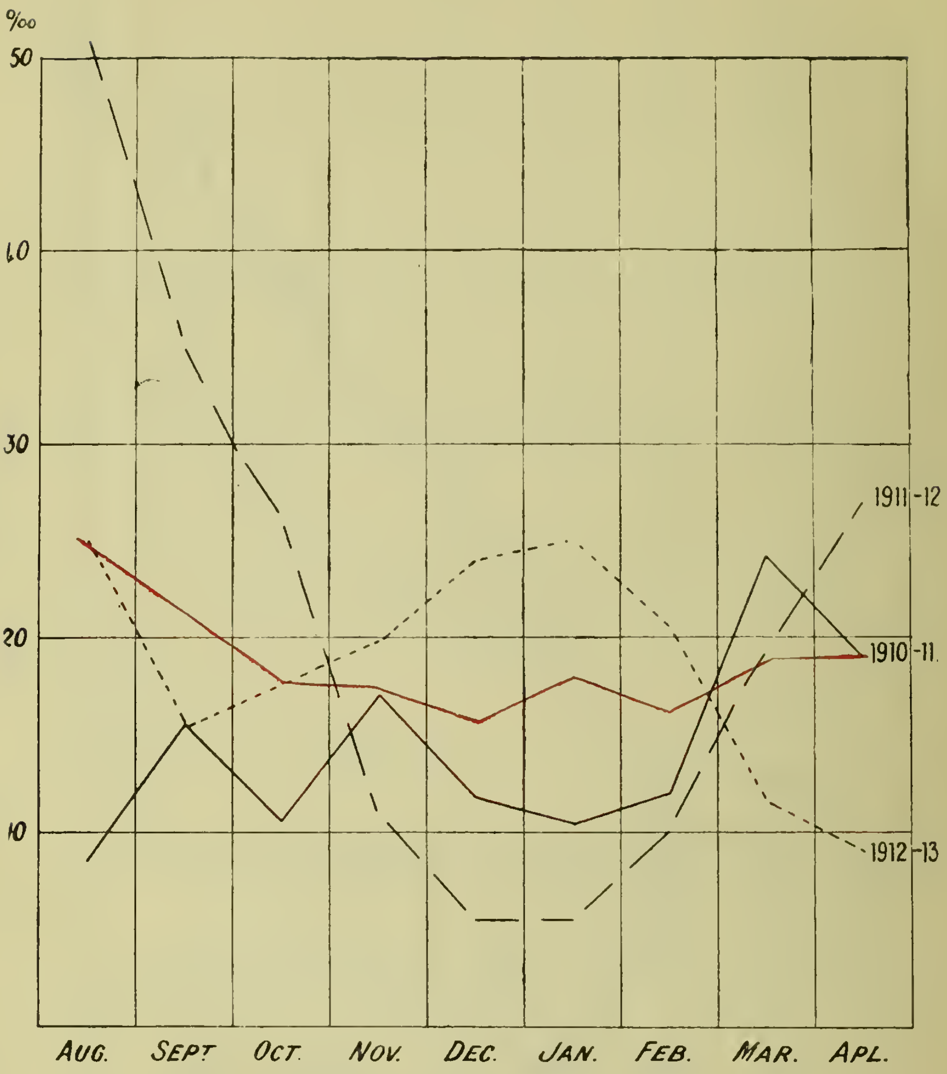


First Curve $y = 54{\cdot}79\left(\frac{x}{{\cdot}7662} - 1\right)^{-{\cdot}1574}\left(1 - \frac{x}{3{\cdot}3791}\right)^{{\cdot}6942}$ $N = 70 = 9{\cdot}8\%$

Second Curve $y = 45{\cdot}43\left(1 + \frac{x}{6{\cdot}8588}\right)^{1{\cdot}0758}\left(1 - \frac{x}{17{\cdot}6066}\right)^{2{\cdot}7616}$ $N = 650 = 90{\cdot}2\%$

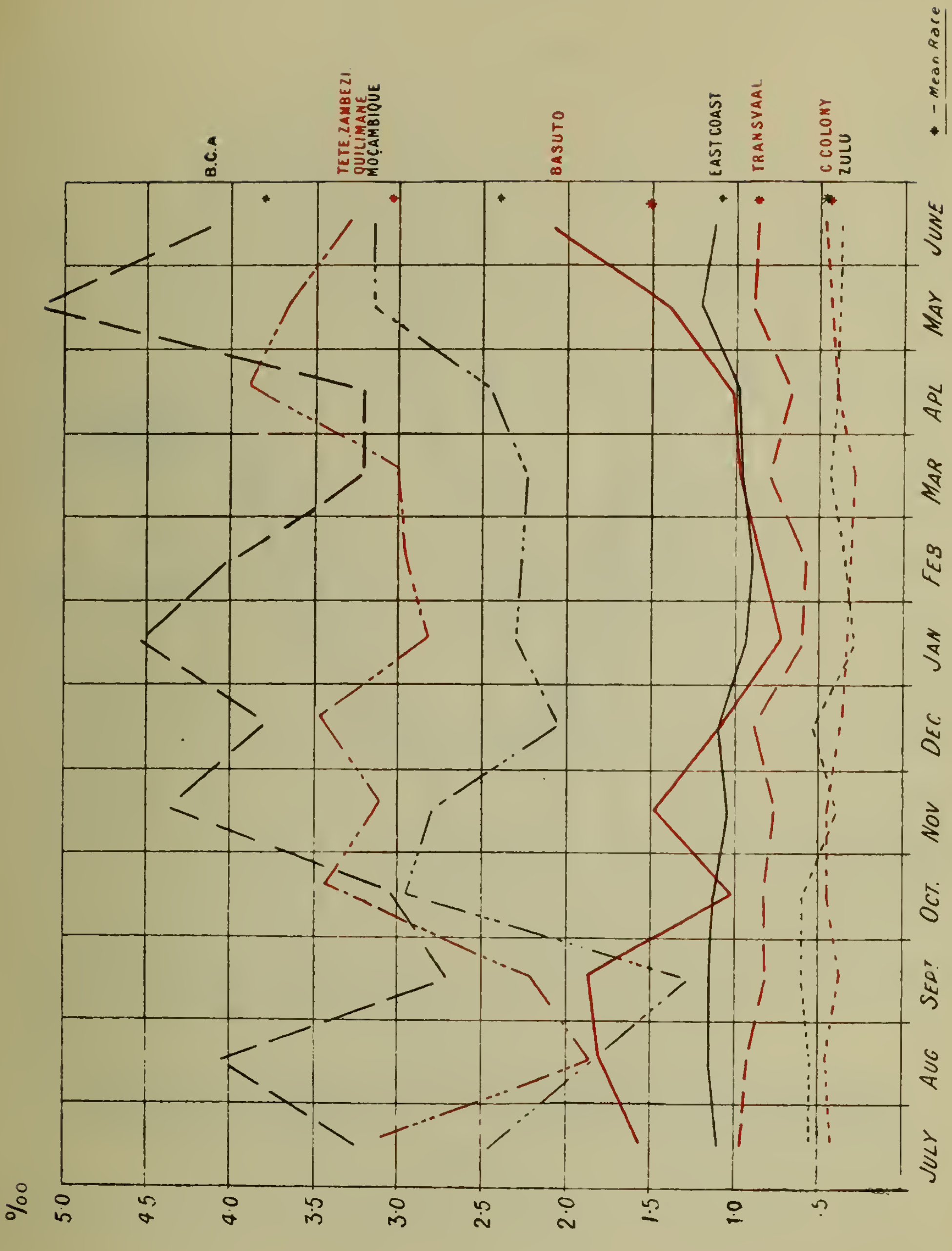
$N = 720.$

CHART IV.



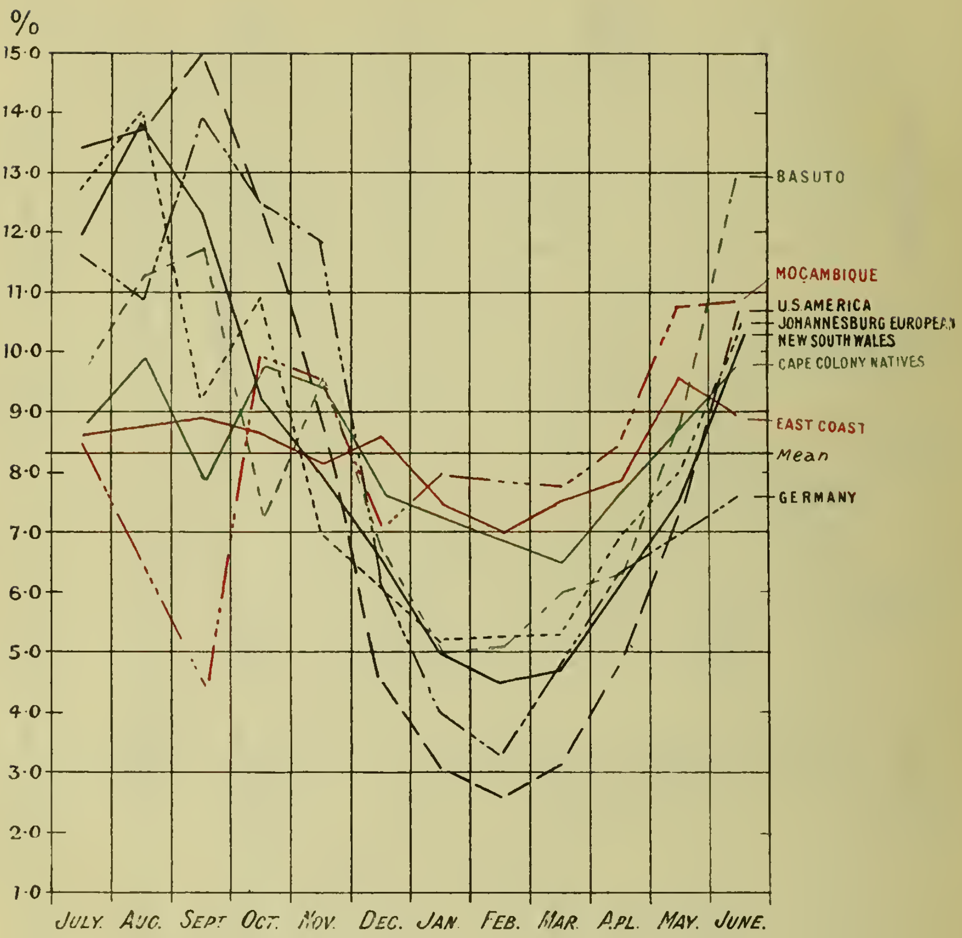
Death-rate from pneumonia for months August—April.
Red line, average of three years.

CHART V.



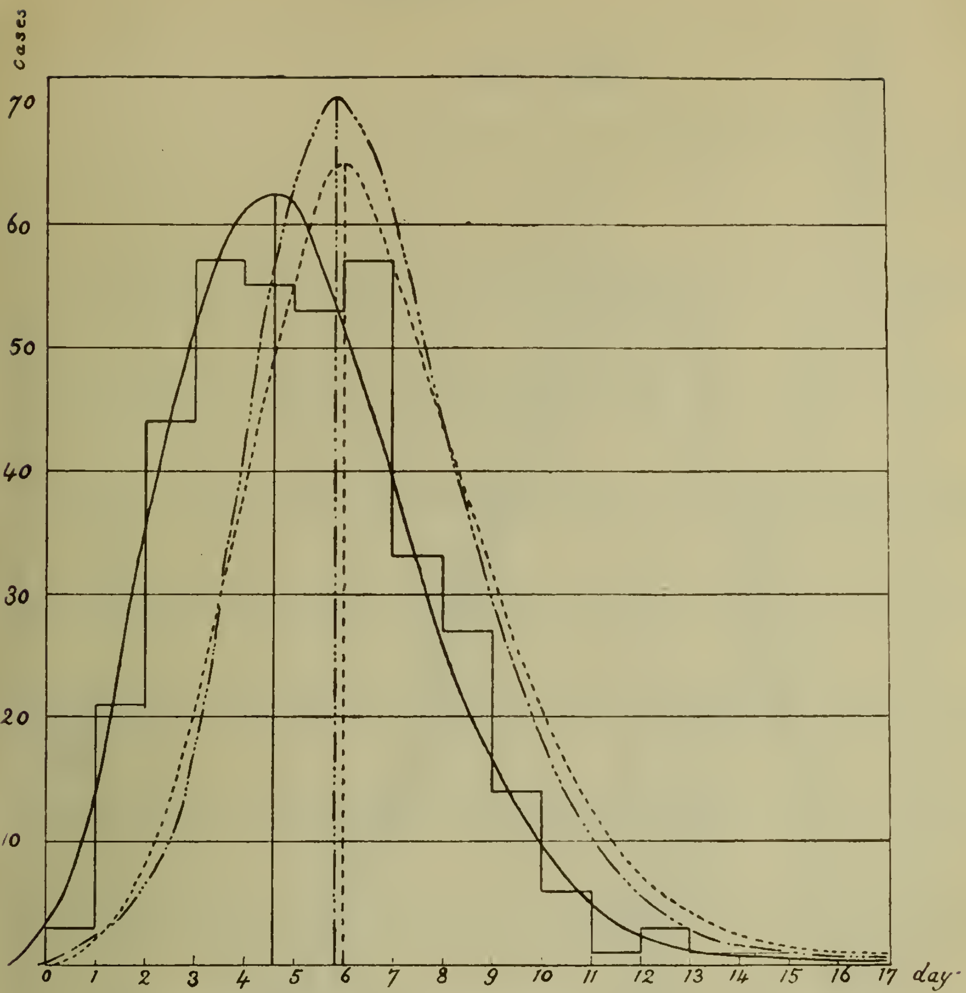
Death-rate per 1,000 for month. Average of five years. 1908-1912.

CHART VI



Percentage of total death-rate occurring in each month. Native figures based on five year average. U.S.A. and German distributions, January read as July, etc.

CHART VII.



Showing day on which crisis occurred in 378 cases of pneumonia amongst Natives from tropical areas, on the Rand. For comparison two curves for distribution of termination by crisis from European figures are given reduced in scale, so that total area of all three curves are the same.

————— = Native data.

—— - - - —— = Musser & Morris 4718 cases.

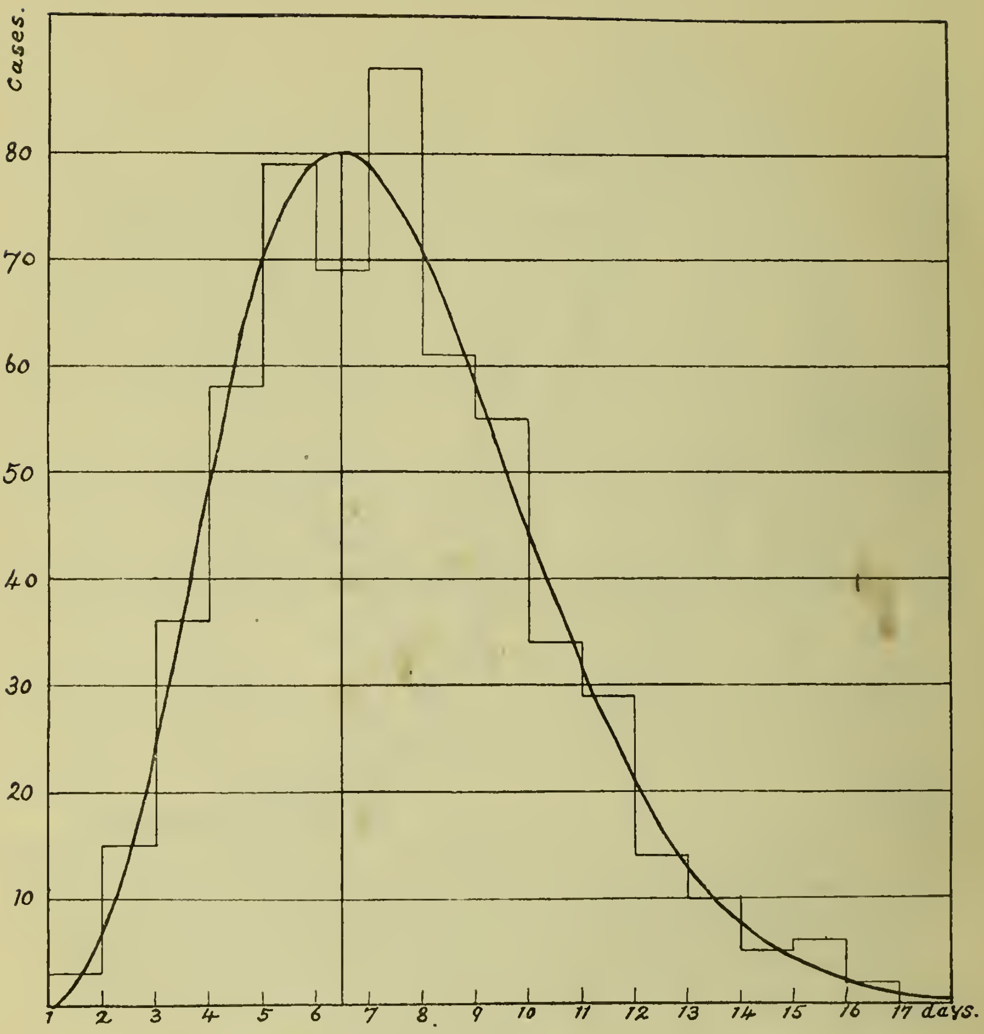
- - - - - - - - - - = Aufrecht 873 cases.

Natives $y = 62{\cdot}26\left(1+\frac{x}{5{\cdot}6590}\right)^{4\cdot2934}\left(1-\frac{x}{25{\cdot}7944}\right)^{19\cdot5700}$

Musser & Morris $y = {\cdot}006108\left(1+\frac{x^2}{40{\cdot}9510}\right)^{-10\cdot9291} e^{24\cdot5806 \tan \frac{x}{6\cdot3993}}$

Aufrecht $y = 4{\cdot}4340\left(1+\frac{x^2}{53{\cdot}3981}\right)^{-7\cdot8771} e^{10\cdot8782 \tan \frac{x}{7\cdot3074}}$

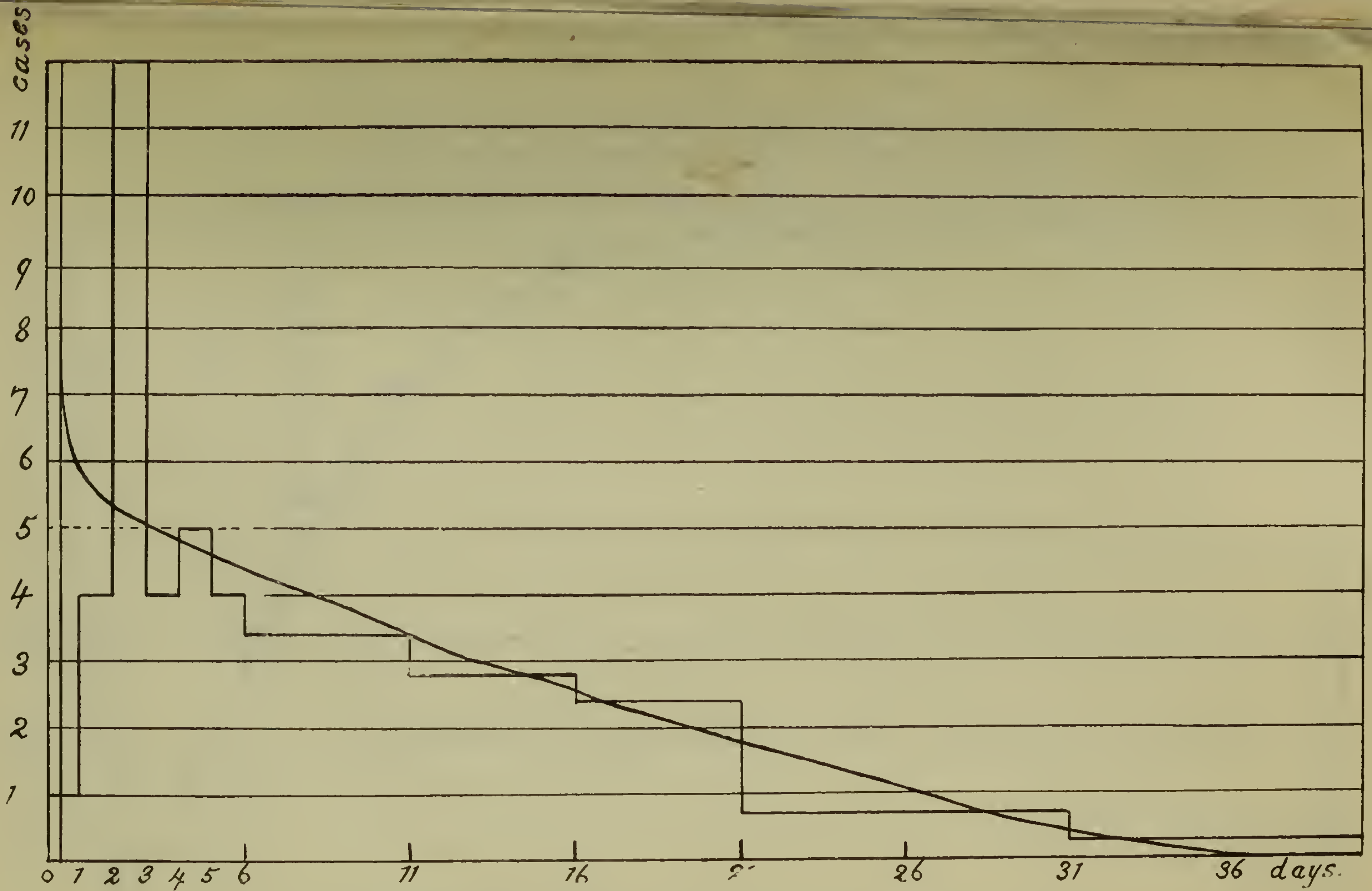
CHART VIII.



Showing day on which the temperature reached the normal by lysis, in 564 cases of pneumonia amongst "Tropical Natives" in W.N.L.A. Hospital.

$$y = 79{\cdot}84 \left(1 + \frac{x}{5{\cdot}8624}\right)^{3{\cdot}2072} \left(1 - \frac{x}{22{\cdot}7921}\right)^{12{\cdot}4691}$$

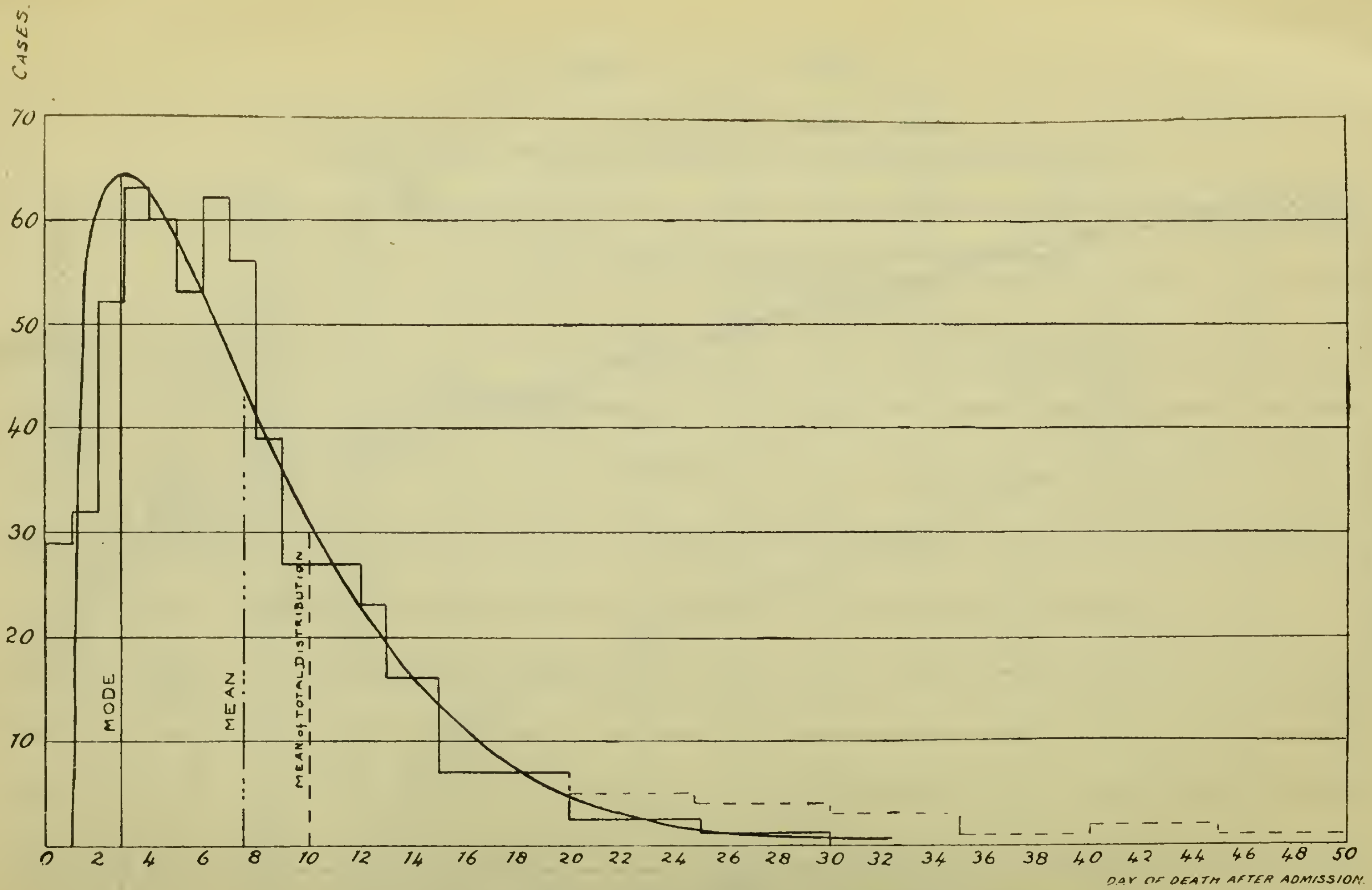
CHART IX.



Second attacks of pneumonia according to number of days after termination of pyrexia of first attack. Cases in W.N.L.A. Compound Hospital.

$$y = 5{\cdot}912\left(\frac{x}{1{\cdot}1691} -\right)^{-{\cdot}0391}\left(1 - \frac{x}{36{\cdot}4804}\right)^{1{\cdot}2201}$$

CHART X.

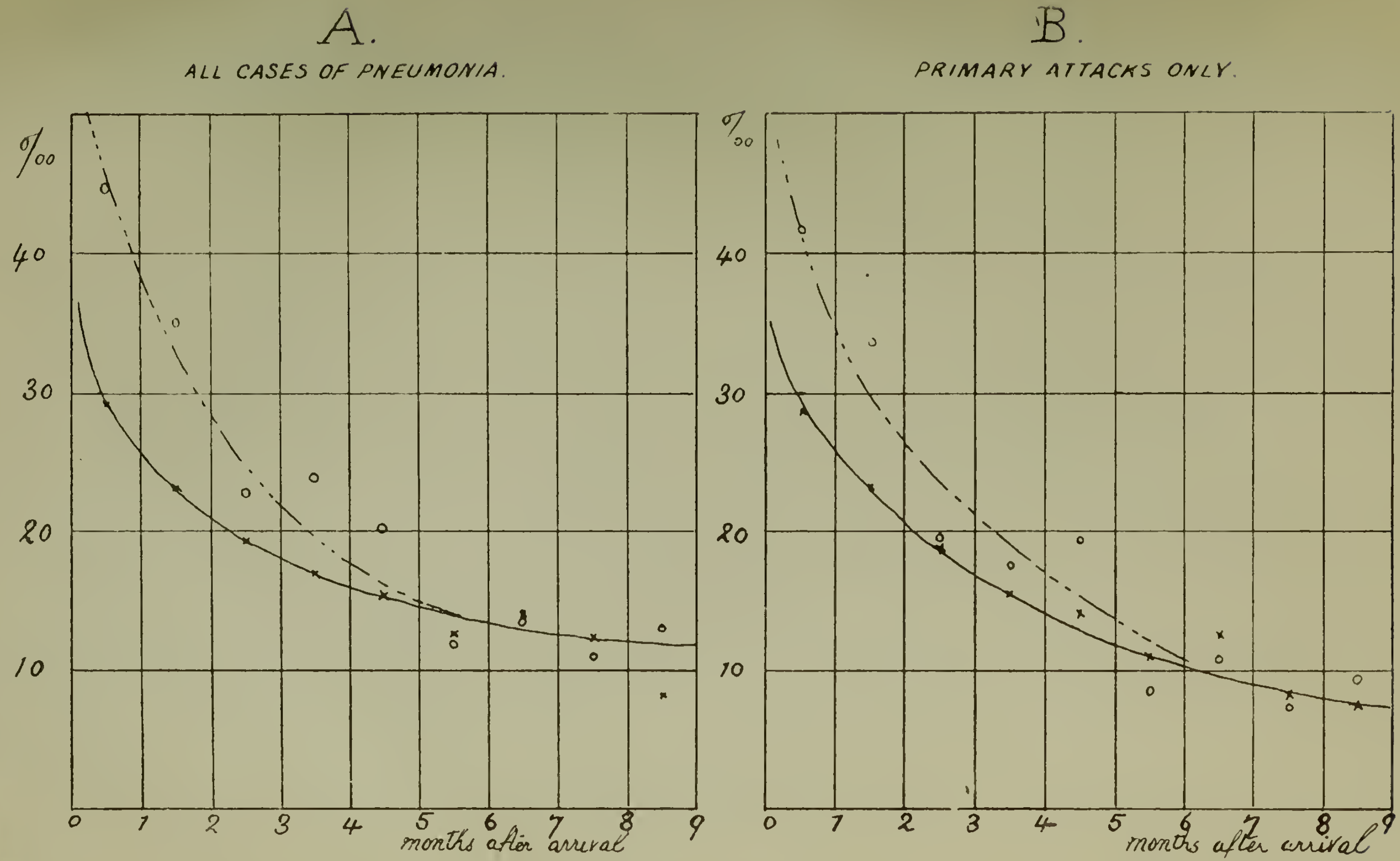


Day of death after admission to W.N.L.A. Hospital. Pneumonia.

All cases $N=705$. J_1 Start 3·0915. End 103·8423 Mean 10·0504.

Dotted tail omitted $N=640$. I. $y=64{\cdot}25\left(1+\frac{x}{2{\cdot}1761}\right)^{{\cdot}3568}\left(1-\frac{x}{54{\cdot}9565}\right)^{9{\cdot}0110}$

CHART XI.



A. ALL CASES OF PNEUMONIA.

B. PRIMARY ATTACKS ONLY.

Attack rates per 1,000, for months after inoculation on arrival at W N.L A Compound.